

10/728766

=> d his

(FILE 'HOME' ENTERED AT 14:18:13 ON 11 APR 2006)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS, LIFESCI' ENTERED AT 14:18:37 ON 11 APR 2006

L1 25776 S THIOREDOXIN  
L2 7634805 S CLON? OR EXPRESS? OR RECOMBINANT  
L3 12217 S L1 AND L2  
L4 7209 S HISTIDINE (W) TAG?  
L5 83 S L3 AND L4  
L6 662353 S DYE?  
L7 0 S L5 AND L6  
L8 48 S L3 AND L6  
L9 37 DUP REM L8 (11 DUPLICATES REMOVED)  
L10 50437 S INCLUSION (W) BOD?  
L11 1 S L9 AND L10  
E CHATTERJEE D K/AU  
L12 550 S E3-E12  
E LONGO M/AU  
L13 698 S E3  
E OBERFELDER R/AU  
L14 56 S E3-E7  
E FLYNN E/AU  
L15 285 S E3  
L16 1578 S L11 OR L12 OR L13 OR L14 OR L15  
L17 1 S L9 AND L16

=>

Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:sssptal652mxm

PASSWORD:

TERMINAL (ENTER 1, 2, 3, OR ?):2

\* \* \* \* \* Welcome to STN International \* \* \* \* \*

NEWS 1 Web Page URLs for STN Seminar Schedule - N. America  
NEWS 2 "Ask CAS" for self-help around the clock  
NEWS 3 DEC 23 New IPC8 SEARCH, DISPLAY, and SELECT fields in USPATFULL/  
USPAT2  
NEWS 4 JAN 13 IPC 8 searching in IFIPAT, IFIUDB, and IFICDB  
NEWS 5 JAN 13 New IPC 8 SEARCH, DISPLAY, and SELECT enhancements added to  
INPADOC  
NEWS 6 JAN 17 Pre-1988 INPI data added to MARPAT  
NEWS 7 JAN 17 IPC 8 in the WPI family of databases including WPIFV  
NEWS 8 JAN 30 Saved answer limit increased  
NEWS 9 FEB 21 STN AnaVist, Version 1.1, lets you share your STN AnaVist  
visualization results  
NEWS 10 FEB 22 The IPC thesaurus added to additional patent databases on STN  
NEWS 11 FEB 22 Updates in EPFULL; IPC 8 enhancements added  
NEWS 12 FEB 27 New STN AnaVist pricing effective March 1, 2006  
NEWS 13 FEB 28 MEDLINE/LMEDLINE reload improves functionality  
NEWS 14 FEB 28 TOXCENTER reloaded with enhancements  
NEWS 15 FEB 28 REGISTRY/ZREGISTRY enhanced with more experimental spectral  
property data  
NEWS 16 MAR 01 INSPEC reloaded and enhanced  
NEWS 17 MAR 03 Updates in PATDPA; addition of IPC 8 data without attributes  
NEWS 18 MAR 08 X.25 communication option no longer available after June 2006  
NEWS 19 MAR 22 EMBASE is now updated on a daily basis  
NEWS 20 APR 03 New IPC 8 fields and IPC thesaurus added to PATDPAFULL  
NEWS 21 APR 03 Bibliographic data updates resume; new IPC 8 fields and IPC  
thesaurus added in PCTFULL  
NEWS 22 APR 04 STN AnaVist \$500 visualization usage credit offered  
  
NEWS EXPRESS FEBRUARY 15 CURRENT VERSION FOR WINDOWS IS V8.01a,  
CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),  
AND CURRENT DISCOVER FILE IS DATED 19 DECEMBER 2005.  
V8.0 AND V8.01 USERS CAN OBTAIN THE UPGRADE TO V8.01a AT  
<http://download.cas.org/express/v8.0-Discover/>  
  
NEWS HOURS STN Operating Hours Plus Help Desk Availability  
NEWS LOGIN Welcome Banner and News Items

Enter NEWS followed by the item number or name to see news on that  
specific topic.

All use of STN is subject to the provisions of the STN Customer  
agreement. Please note that this agreement limits use to scientific  
research. Use for software development or design or implementation  
of commercial gateways or other similar uses is prohibited and may  
result in loss of user privileges and other penalties.

\* \* \* \* \* STN Columbus \* \* \* \* \*

FILE 'HOME' ENTERED AT 14:18:13 ON 11 APR 2006

=> file medline embase biosis biotechds scisearch hcaplus ntis lifesci		
COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'MEDLINE' ENTERED AT 14:18:37 ON 11 APR 2006

FILE 'EMBASE' ENTERED AT 14:18:37 ON 11 APR 2006  
Copyright (c) 2006 Elsevier B.V. All rights reserved.

FILE 'BIOSIS' ENTERED AT 14:18:37 ON 11 APR 2006  
Copyright (c) 2006 The Thomson Corporation

FILE 'BIOTECHDS' ENTERED AT 14:18:37 ON 11 APR 2006  
COPYRIGHT (C) 2006 THE THOMSON CORPORATION

FILE 'SCISEARCH' ENTERED AT 14:18:37 ON 11 APR 2006  
Copyright (c) 2006 The Thomson Corporation

FILE 'HCAPLUS' ENTERED AT 14:18:37 ON 11 APR 2006  
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.  
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.  
COPYRIGHT (C) 2006 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'NTIS' ENTERED AT 14:18:37 ON 11 APR 2006  
Compiled and distributed by the NTIS, U.S. Department of Commerce.  
It contains copyrighted material.  
All rights reserved. (2006)

FILE 'LIFESCI' ENTERED AT 14:18:37 ON 11 APR 2006  
COPYRIGHT (C) 2006 Cambridge Scientific Abstracts (CSA)

=> s thioredoxin  
L1 25776 THIOREDOXIN

=> s clon? or express? or recombinant  
L2 7634805 CLON? OR EXPRESS? OR RECOMBINANT

=> s l1 and l2  
L3 12217 L1 AND L2

=> s histidine (w) tag?  
L4 7209 HISTIDINE (W) TAG?

=> s l3 and l4  
L5 83 L3 AND L4

=> s dye?  
L6 662353 DYE?

=> s l5 and l6  
L7 0 L5 AND L6

=> s l3 and l6  
L8 48 L3 AND L6

=> dup rem l8  
PROCESSING COMPLETED FOR L8  
L9 37 DUP REM L8 (11 DUPLICATES REMOVED)

=> d 1-37 ibib ab

L9 ANSWER 1 OF 37 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2006137243 EMBASE  
TITLE: Database of two-dimensional polyacrylamide gel electrophoresis of proteins labeled with CyDye DIGE Fluor saturation dye.  
AUTHOR: Fujii K.; Kondo T.; Yokoo H.; Okano T.; Yamada M.; Yamada T.; Iwatsuki K.; Hirohashi S.  
CORPORATE SOURCE: Prof. T. Kondo, Cancer Proteomics Project, National Cancer Center Research Institute, 5-1-1 Tsukiji, Chuo-ku, Tokyo 104-0045, Japan. takondo@gan2.res.ncc.go.jp  
SOURCE: Proteomics, (2006) Vol. 6, No. 5, pp. 1640-1653. .  
Refs: 27  
ISSN: 1615-9853 E-ISSN: 1615-9861 CODEN: PROTC7  
COUNTRY: Germany  
DOCUMENT TYPE: Journal; Article  
FILE SEGMENT: 016 Cancer  
029 Clinical Biochemistry  
048 Gastroenterology  
LANGUAGE: English  
SUMMARY LANGUAGE: English  
ENTRY DATE: Entered STN: 5 Apr 2006  
Last Updated on STN: 5 Apr 2006

AB CyDye DIGE Fluor saturation dye (saturation dye, GE Healthcare Amersham Biosciences) enables highly sensitive 2-D PAGE. As the dye reacts with all reduced cysteine thiols, 2-D PAGE can be performed with a lower amount of protein, compared with CyDye DIGE Fluor minimal dye (GE Healthcare Amersham Biosciences), the sensitivity of which is equivalent to that of silver staining. We constructed a 2-D map of the saturation dye-labeled proteins of a liver cancer cell line (HepG2) and identified by MS 92 proteins corresponding to 123 protein spots. Functional classification revealed that the identified proteins had chaperone, protein binding, nucleotide binding, metal ion binding, isomerase activity, and motor activity. The functional distribution and the cysteine contents of the proteins were similar to those in the most comprehensive 2-D database of hepatoma cells (Seow et al., Electrophoresis 2000, 21, 1787-1813), where silver staining was used for protein visualization. Hierarchical clustering on the basis of the quantitative expression profiles of the 123 characterized spots labeled with two charge- and mass-matched saturation dyes (Cy3 and Cy5) discriminated between nine hepatocellular carcinoma cell lines and primary cultured hepatocytes from five individuals, suggesting the utility of saturation dye and our database for proteomic studies of liver cancer. .COPYRGHT. 2006 Wiley-VCH Verlag GmbH & Co. KGaA.

L9 ANSWER 2 OF 37 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2005:1311323 HCAPLUS  
DOCUMENT NUMBER: 144:47000  
TITLE: Lung endothelial cell associated marker proteins as targets for tissue-specific imaging and therapeutical agents in diagnosis and therapy  
INVENTOR(S): Schnitzer, Jan E.; Oh, Phillip  
PATENT ASSIGNEE(S): Sidney Kimmel Cancer Center, USA  
SOURCE: PCT Int. Appl., 84 pp.  
CODEN: PIXXD2  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 1  
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005117977	A2	20051215	WO 2005-US19398	20050602

WO 2005117977 A3 20060202

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2006024231 A1 20060202 US 2005-143114 20050602

PRIORITY APPLN. INFO.: US 2004-576114P P 20040602

AB Methods of delivering an agent in a tissue-specific manner, particularly lung tissue, by targeting a protein **expressed** on the endothelial cell surface, are described. The methods can be used for detecting, imaging and/or treating pathologies, as well as for diagnostics. Specifically claimed are a series of lung endothelial cell associated marker proteins for diagnostic and therapeutical uses, in particular TIE-2, APN, TEM4, TEM6, ICAM-1, nucleolin, P2Z receptor, Trk-A, FLJ10849, HSPA12B, APP, and OX-45.

L9 ANSWER 3 OF 37 MEDLINE on STN

ACCESSION NUMBER: 2005629887 MEDLINE

DOCUMENT NUMBER: PubMed ID: 16159878

TITLE: Endogenous **thioredoxin** is required for redox cycling of anthracyclines and p53-dependent apoptosis in cancer cells.

AUTHOR: Ravi Dashnamoorthy; Muniyappa Harish; Das Kumuda C  
CORPORATE SOURCE: Department of Pathology and Arkansas Cancer Research Center, University of Arkansas for Medical Sciences, Little Rock, Arkansas 72205, USA.

CONTRACT NUMBER: 1R01HL071558 (NHLBI)

SOURCE: The Journal of biological chemistry, (2005 Dec 2) Vol. 280, No. 48, pp. 40084-96. Electronic Publication: 2005-09-13. Journal code: 2985121R. ISSN: 0021-9258.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200602

ENTRY DATE: Entered STN: 20051129

Last Updated on STN: 20060204

Entered Medline: 20060203

AB Apoptosis is a major mechanism of cancer cell destruction by chemotherapy and radiotherapy. The anthracycline class of antitumor drugs undergoes redox cycling in living cells producing increased amounts of reactive oxygen species and semiquinone radical, both of which can cause DNA damage, and consequently trigger apoptotic death of cancer cells. We show here that MCF-7 cells overexpressing **thioredoxin** (Trx) were more apoptotic in response to daunomycin. Trx overexpression in MCF-7 cells increased the generation of superoxide anion (O<sub>2</sub><sup>•-</sup>) in anthracycline-treated cell extracts. Enhanced generation of O<sub>2</sub><sup>•-</sup> in response to daunomycin in Trx-overexpressing MCF-7 cells was inhibited by diphenyleneiodonium chloride, a general NADPH reductase inhibitor, demonstrating that Trx provides reducing equivalents to a bioreductive enzyme for redox cycling of daunomycin. Additionally Trx increased p53-DNA binding and **expression** in response to anthracyclines. MCF-7 cells **expressing** mutant redox-inactive Trx showed decreased superoxide generation, apoptosis, and p53 protein and DNA binding. In addition, down-regulation of endogenous Trx **expression** by small interfering RNA resulted in decreased

expression of caspase-7 and cleaved poly(ADP-ribose) polymerase expression in response to daunomycin. These results suggest that endogenous Trx is required for anthracycline-mediated apoptosis of breast cancer cells. Taken together, our data demonstrate a novel pro-oxidant and proapoptotic role of Trx in anthracycline-mediated apoptosis in anthracycline chemotherapy.

L9 ANSWER 4 OF 37 MEDLINE on STN  
ACCESSION NUMBER: 2005018546 MEDLINE  
DOCUMENT NUMBER: PubMed ID: 15643880  
TITLE: Synthesis and characterization of dimaleimide fluorogens designed for specific labeling of proteins.  
AUTHOR: Girouard Stephane; Houle Marie-Helene; Grandbois Alain; Keillor Jeffrey W; Michnick Stephen W  
CORPORATE SOURCE: Department of Chemistry, Universite de Montreal, C.P. 6128, Succ. Centre-Ville, Montreal, Quebec H3C 3J7 Canada.  
SOURCE: Journal of the American Chemical Society, (2005 Jan 19) Vol. 127, No. 2, pp. 559-66.  
Journal code: 7503056. ISSN: 0002-7863.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 200503  
ENTRY DATE: Entered STN: 20050113  
Last Updated on STN: 20050304  
Entered Medline: 20050303

AB A series of aromatic compounds were prepared bearing two maleimide groups attached directly to the fluorescent cores. The resulting derivatives do not fluoresce until the maleimide groups undergo their typical thiol addition reaction, thus removing their ability to quench fluorescence, as shown by kinetic and spectral characterization studies. In this way, the title compounds serve as fluorogens capable of detection of small thiols or appropriately sized dithiols. Recombinant alpha-helical proteins were then designed to bear two cysteine residues capable of regioselective dithiol addition reaction with the dimaleimide fluorogens, thus acting as spatially encoded substrates that form specifically labeled covalent complexes. The efficiency of this in vitro fluorescent protein-labeling reaction demonstrates the feasibility of the development of a method for the fluorescent labeling of specific recombinant proteins.

L9 ANSWER 5 OF 37 HCAPLUS COPYRIGHT 2006 ACS on STN  
ACCESSION NUMBER: 2005:388294 HCAPLUS  
DOCUMENT NUMBER: 144:65239  
TITLE: Prediction of Nephrotoxicant Action and Identification of Candidate Toxicity-Related Biomarkers  
AUTHOR(S): Thukral, Sushil K.; Nordone, Paul J.; Hu, Rong; Sullivan, Leah; Galambos, Eric; Fitzpatrick, Vincent D.; Healy, Laura; Bass, Michael B.; Cosenza, Mary E.; Afshari, Cynthia A.  
CORPORATE SOURCE: Amgen Inc., Thousand Oaks, CA, 91320, USA  
SOURCE: Toxicologic Pathology (2005), 33(3), 343-355  
CODEN: TOPADD; ISSN: 0192-6233  
PUBLISHER: Taylor & Francis, Inc.  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB A vast majority of pharmacol. compds. and their metabolites are excreted via the urine, and within the complex structure of the kidney, the proximal tubules are a main target site of nephrotoxic compds. The authors used the model nephrotoxicants mercuric chloride, 2-bromoethylamine hydrobromide, hexachlorobutadiene, mitomycin, amphotericin, and puromycin to elucidate time- and dose-dependent global gene expression changes associated with proximal tubular toxicity.

Male Sprague-Dawley rats were dosed via i.p. injection once daily for mercuric chloride and amphotericin (up to 7 doses), while a single dose was given for all other compds. Animals were exposed to 2 different doses of these compds. and kidney tissues were collected on day 1, 3, and 7 postdosing. Gene expression profiles were generated from kidney RNA using 17K rat cDNA dual dye microarray and analyzed in conjunction with histopathol. Anal. of gene expression profiles showed that the profiles clustered based on similarities in the severity and type of pathol. of individual animals. Further, the expression changes were indicative of tubular toxicity showing hallmarks of tubular degeneration/regeneration and necrosis. Use of gene expression data in predicting the type of nephrotoxicity was then tested with a support vector machine (SVM)-based approach. A SVM prediction module was trained using 120 profiles of total profiles divided into four classes based on the severity of pathol. and clustering. Although mitomycin C and amphotericin B treatments did not cause toxicity, their expression profiles were included in the SVM prediction module to increase the sample size. Using this classifier, the SVM predicted the type of pathol. of 28 test profiles with 100% selectivity and 82% sensitivity. These data indicate that valid predictions could be made based on gene expression changes from a small set of expression profiles. A set of potential biomarkers showing a time- and dose-response with respect to the progression of proximal tubular toxicity were identified. These include several transporters (Slc21a2, Slc15, Slc34a2), Kim 1, IGFbp-1, osteopontin, a -fibrinogen, and Gsta.

REFERENCE COUNT: 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 6 OF 37 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2005:1115101 HCAPLUS

DOCUMENT NUMBER: 143:433934

TITLE: Gene expression profiling of responses to dimethylarsinic acid in female F344 rat urothelium

AUTHOR(S): Sen, Banalata; Wang, Amy; Hester, Susan D.; Robertson, John L.; Wolf, Douglas C.

CORPORATE SOURCE: Environmental Carcinogenesis Division, National Health and Environmental Effects Laboratory, US Environmental Protection Agency, Research Triangle Park, NC, 27711, USA

SOURCE: Toxicology (2005), 215(3), 214-226

CODEN: TXCYAC; ISSN: 0300-483X

PUBLISHER: Elsevier Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Gene expression profiling has been shown to be useful for identifying underlying mechanisms of toxicity, determining patterns of biol. response, and elucidating candidate markers of exposure and response. Inorg. arsenic (iAs) is a human carcinogen and epidemiol. evidence implicates it in the development of urinary bladder cancer. Dimethylarsinic acid (DMA), the major excreted metabolite of iAs in humans, is a known rat bladder carcinogen. To examine the changes associated with DMA exposure, microarray anal. of the urothelium was performed in female F344 rats exposed to non-toxic and toxic doses of DMA in their drinking water for 28 days. A novel method for isolating predominantly urothelial cells was developed. Gene expression profiling of the urothelium using a custom 2-dye spotted array revealed that DMA treatment modulated the expression of transcripts of genes that regulate apoptosis, cell cycle regulation and the oxidative stress response. Expression of genes mapping to pathways involved in cancer control processes were also altered after DMA exposure. Morphol. data suggested a dose dependent increase in cellular toxicity. Significant changes in differential gene expression were present after all treatments event at doses where standard toxicol. responses were not

detectable. The greatest perturbation in gene expression was present in rats after treatment with 40 ppm DMA. Doses which produced no histol. or ultrastructural evidence of toxicity (non-toxic) could be differentiated from toxic doses based on the expression of a subset of genes, which control cell signaling and the stress response. These reported changes in gene expression show similarities between the mechanisms of action of DMA in vivo and those previously described for iAs in vitro. These data illustrate the utility of transcriptional profiling and its potential in predicting key mechanistic pathways involved in toxicity and as a time efficient tool to inform the mode of action anal. in risk assessment.

REFERENCE COUNT: 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 7 OF 37 BIOTECHDS COPYRIGHT 2006 THE THOMSON CORP. on STN  
ACCESSION NUMBER: 2004-13947 BIOTECHDS

TITLE: Novel polypeptide antagonist to peripheral-type benzodiazepine receptor, useful for treating disease or condition such as breast cancer, multiple sclerosis, cirrhosis, ischemia and Alzheimer's disease; antagonist against receptor for use in disease therapy and gene therapy

AUTHOR: PAPADOPOULOS V; GAZOULI M

PATENT ASSIGNEE: UNIV GEORGETOWN

PATENT INFO: WO 2004031722 15 Apr 2004

APPLICATION INFO: WO 2003-US29822 23 Sep 2003

PRIORITY INFO: US 2002-414635 1 Oct 2002; US 2002-414635 1 Oct 2002

DOCUMENT TYPE: Patent

LANGUAGE: English

OTHER SOURCE: WPI: 2004-348000 [32]

AB DERWENT ABSTRACT:

NOVELTY - A polypeptide antagonist (I) to the peripheral-type benzodiazepine receptor (PBR), is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for: (1) identifying (M1) an antagonist to the PBR, involving: (a) obtaining a library comprising random polypeptides; (b) screening the library to identify polypeptides that bind to PBR; and (c) testing the identified polypeptides for the ability to prevent binding of a recognized PBR ligand, where the identified polypeptide that prevents binding of a PBR ligand is an antagonist of PBR; (2) an antagonist (II) of the PBR identified by (M1); (3) screening (M2) for compounds which are antagonists to the PBR receptor, involving: (a) attaching PBR or its biologically-active polypeptide fragment to a substrate; (b) exposing the PBR or its biologically-active polypeptide fragment to one or more compounds; (c) determining whether the compound is bound to the PBR or its biologically-active polypeptide fragment; and (d) if the compound is bound, further determining whether the compound modulates the interaction between the PBR ligand and the PBR; (4) a compound identified by (M2); (5) a composition (III) for treating a PBR-related disease or condition, comprising a compound which acts as an antagonist to the PBR; (6) preparing (III), involves combining an antagonist to the PBR and a carrier; (7) a kit (IV) comprising an antagonist to the PBR; (8) a compound identified using (II); (9) an isolated polypeptide (V) comprising a motif (MF) Ser-Thr-XXXX-Pro, where the polypeptide modulates PBR and X represents any amino acid; (10) an antibody (VI) or its biologically-active fragment which recognizes and binds to PBR antagonist amino acid sequence which contains MF; (11) a nucleic acid (VII) encoding (I); (12) a vector comprising (VII); and (13) an antagonist of the PBR which is not a polypeptide, where the agonist is a peptide mimetic designed to correspond to a polypeptide antagonist of PBR.

WIDER DISCLOSURE - Detecting the ability of a test sample to affect the binding interaction of a first polypeptide and a second polypeptide of a polypeptide binding pair, where the first polypeptide is a PBR antagonist polypeptide and the second polypeptide is PBR is also



disclosed.

**BIOTECHNOLOGY** - Preferred Antagonist: (I) blocks agonists of the PBR by competitive binding at the ligand binding site of the PBR, where the polypeptide contains MF, and X represents any amino acid. The polypeptide comprises an amino acid sequence (S1) Ser-Thr-Pro-His-Ser-Thr-Pro, which is linked to the TAT sequence derived from HIV. Preferred Kit: In (IV), the antagonists comprises a polypeptide which contains MF. The polypeptide comprises (S1). Preferred Antibody: In (VI), the amino acid sequence comprises (S1). Preferred Polypeptide: (V) comprises (S1), and further comprises TAT sequence of HIV. Preferred Nucleic Acid: In (VII), the polypeptide antagonist is an aptamer, comprising a nucleic acid encoding a scaffold protein in-frame with the polypeptide aptamer. The scaffold protein is thioredoxin A.

**ACTIVITY** - Cytostatic; Neuroprotective; Tranquilizer; Antidepressant; Nootropic; Hepatotropic; Vasotropic; Antiparkinsonian; Vulnerary.

**MECHANISM OF ACTION** - Antagonist of PBR; Blocks agonist of PBR by competitive binding at the ligand binding site of the PBR; Regulator of PBR activity (claimed). In vitro analysis of PBR antagonist in inhibiting steroid biosynthesis, was carried out as follows: MA-10 Leydig cells were incubated in the presence of increasing concentrations of TAT-Ser-Thr-Pro-His-Ser-Thr-Pro for 30 minutes. Cells were then exposed to 1 microm of Ro5-4864 (4'-chlorodiazepam). After 4 hours, steroid secreted into the media, was measured by radioimmunoassay. The result indicated inhibition of steroid formation by the sequence with an ED50 of 5 microm. Thus, the sequence was found to act as a competitive PBR antagonist.

**USE** - (I) is useful for treating a PBR-related disease or condition in a subject such as canine, feline, ovine, equine, porcine, caprine, camelid, avian, bovine, amphibian, fish, murine or primate organism such as human, which involves administering (I) to the subject, where the PBR-related disease or condition is chosen from breast cancer, colon cancer, prostate cancer, multiple sclerosis, alcohol withdrawal, affective disorders, anxiety disorders, seizures, brain tumors, Alzheimer's disease, stress, acute myeloid leukemia, liver cancer, cirrhosis, traumatic brain injury, ischemia, reactions associated with chemical toxins, and their combinations. (I) is useful for regulating a PBR activity in vivo, which involves administering (I), where (I) modulates PBR by binding with the ligand binding site of PBR. (II) is useful for identifying one or more agents that modulate PBR ligand binding, which involves binding a PBR or a ligand binding domain of PBR to a solid support, adding (II) to the PBR or its fragment which binds to PBR in the presence of the agent, measuring the amount of the ligand binding domain of PBR bound to (II) on the support in the presence of the agent, and identifying an agent that reduces the amount of the ligand binding domain of PBR binding to (II) on the support, where the solid substrate is a microarray. (III) is useful for treating the above-mentioned diseases (claimed). (I) is useful for treating disease or condition such as Parkinson's disease and ovarian cancer.

**ADMINISTRATION** - (I) is administered by inhalation, subcutaneous, intramuscular, intravenous injection, or by oral route. No specific dosage details are given.

**EXAMPLE** - Purified human chorionic gonadotrophin (hCG) was obtained. MA-10 cells were grown in modified Waymouth's MB752/1 medium containing 15% horse serum. Mitochondria were isolated. A polypeptide library comprising  $1.28 \times 10^9$  to the power of 9 possible 7-mer sequences, were obtained. As protein target, MA-10 isolated mitochondria were used. In brief, a microtiter well was coated overnight with 150 microl of 100 microg/ml MA-10 mitochondria in 1X phosphate buffered saline (PBS), using 0.1 M sodium bicarbonate (pH of 8.6) and blocked with a blocking solution containing 0.1 M sodium bicarbonate (pH of 8.6), 5 mg/ml bovine serum albumin (BSA) and 0.02% sodium nitrite. Peripheral-type benzodiazepine receptor (PBR) function on coated mitochondria was examined using radioligand binding assays. About  $2 \times 10^9$  transducing units of phage from

the library were combined and incubated for 1 hour with the coated well at room temperature in 100 microl TBST buffer (50 mM Tris-hydrochloric acid (pH of 7.6), 150 mM sodium chloride and 0.1% (v/v) Tween-20). Wells were washed 10 times with TBST and eluted with 0.1 M of 1-(2-chlorophenyl)-N-methyl-N- (1-methyl-propyl)-3-isoquinoline carboxamide (PK 11195). DNA from individual phage clones was sequenced after three rounds of selection. Single-stranded DNA from individual phage clones was purified. Nucleotide sequences were analyzed on a DNA sequencer using the sequencing primer having sequence of 5'-HO-CCCTCATAGTTAGCGTAACG-3'. Radioligand binding assays were performed. MA-10 mitochondria were suspended in 1X PBS at a final concentration of 10 microg protein/100 microl. Specific radioligand binding (2.5 nM) to MA-10 mitochondria was measured in the presence or absence of increasing concentrations (10 to the power of -6 M to 10 to the power of -3 M) of competing PBR peptides. In all cases after 120 minutes of incubation at 4 degrees C, the incubation reaction was stopped by filtration through filters. Bound radioactivity was determined by liquid scintillation spectrometry. Twenty-mer TAT-Ser-Thr-Pro-His-Ser-Thr-Pro and TAT-Ser-Thr-His-Glu-Glu-Thr-Pro peptides were synthesized that contained an NH2-terminal 11-mer TAT protein transduction domain, followed by two glycine residues. Transduction experiments were performed. To determine the efficiency of TAT polypeptide incorporation into the cells, MA-10 cells were cultured overnight on 8-chambered Super Cell Culture Slides at a concentration of approximately 25000 cells/chamber. Media were replaced 24 hours later with fresh media and cells were treated with various concentrations of Oregon Green 488-labeled peptides for various time periods. After the incubation period, cells were washed with PBS and examined by fluorescent microscopy. For steroid synthesis experiments, MA-10 cells were plated into 96-well plate at the density of 2.5 x 10 to the power of 4 cells/well. About 24 hours later, media was replaced with fresh media and cells were treated with the indicated concentrations of peptides for 30 minutes. Cells were then stimulated with 50 ng/ml hCG or 20 microM of the hydrosoluble analog of cholesterol, 22 R-hydrocholesterol in serum-free media for 2 hours. At the end of the incubation, culture media were collected and tested for progesterone production by radioimmunoassay using anti-progesterone antisera. Progesterone production was normalized against the amount of protein in each well. Proteins were quantified using dye-binding assay with bovine serum albumin as standard. In biopanning experiments, PBR interacting phage peptides were eluted using PK 11195 or Ro5-4864 (4'-chlorodiazepam). After three rounds of panning, 20 individual polypeptide clones from each eluate were selected, amplified and sequenced. The polypeptide sequences were found to comprise a motif Ser-Thr-XXXX-Pro. (31 pages)

L9 ANSWER 8 OF 37 MEDLINE on STN  
 ACCESSION NUMBER: 2004513579 MEDLINE  
 DOCUMENT NUMBER: PubMed ID: 15378754  
 TITLE: Action and reaction: Chlamydomonas reinhardtii proteome alteration in a persistent infection induced by iron deficiency.  
 AUTHOR: Wehrli Wolfgang; Meyer Thomas F; Jungblut Peter R; Muller Eva-Christina; Szczepiek Agnes J  
 CORPORATE SOURCE: Department of Molecular Biology, Max-Planck Institute for Infection Biology, Berlin, Germany.  
 SOURCE: Proteomics, (2004 Oct) Vol. 4, No. 10, pp. 2969-81. Journal code: 101092707. ISSN: 1615-9853.  
 PUB. COUNTRY: Germany: Germany, Federal Republic of  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 200503  
 ENTRY DATE: Entered STN: 20041015  
 Last Updated on STN: 20050303

Entered Medline: 20050302

AB Chlamydomphila pneumoniae is an obligate intracellular pathogen implicated in a variety of acute and chronic diseases. Long-term infections are associated with a persistent life stage, in which bacteria can stay for years. They are less accessible to antibiotic treatment but still prone to sustain an inflammatory response. Different in vitro models have been established to mimic and characterize chlamydial persistency. For C. pneumoniae and Chlamydia trachomatis, altered metabolic activities and changed antigenic profiles compared to acute infections have been reported. Most studies including transcriptome and proteome analyses describe persistency induced by IFNgamma treatment. Here, we use iron depletion of the infected cell culture that also leads into persistent infection. We describe differently regulated proteins found by subtractive proteome analysis comparing two early stages of infection with and without addition of the iron chelator deferoxamine-mesylate. While only one bacterial protein was up-regulated during iron deficiency up to 24 h post infection (p.i.), 11 were found to be up-regulated and eight to be down-regulated from 24-48 h p.i. Two down-regulated proteins could be identified by peptide mass fingerprinting as thioredoxin reductase and chromosome partitioning protein (ParB). The latter is involved in chromosome segregation. Thus, using a comparative approach we identified on a proteome level down-regulation of ParB in persistent chlamydial forms, which is in agreement with previous results describing changes in cell division and atypical altered morphology of persistent Chlamydiae.

L9 ANSWER 9 OF 37 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2004:446076 SCISEARCH

THE GENUINE ARTICLE: 818YM

TITLE: Oxidant-injured airway epithelial cells upregulate thioredoxin but do not produce interleukin-8

AUTHOR: Oslund K L; Miller L A; Usachenko J L; Tyler N K; Wu R; Hyde D M (Reprint)

CORPORATE SOURCE: Univ Calif Davis, Sch Vet Med, Ctr Comparat Resp Biol & Med, 1 Shields Ave, Davis, CA 95616 USA (Reprint); Univ Calif Davis, Sch Vet Med, Ctr Comparat Resp Biol & Med, Davis, CA 95616 USA; Univ Calif Davis, Sch Vet Med, Dept Anat Physiol & Cell Biol, Davis, CA 95616 USA

COUNTRY OF AUTHOR: USA

SOURCE: AMERICAN JOURNAL OF RESPIRATORY CELL AND MOLECULAR BIOLOGY (MAY 2004) Vol. 30, No. 5, pp. 597-604.  
ISSN: 1044-1549.

PUBLISHER: AMER THORACIC SOC, 1740 BROADWAY, NEW YORK, NY 10019-4374 USA.

DOCUMENT TYPE: Article; Journal

LANGUAGE: English

REFERENCE COUNT: 29

ENTRY DATE: Entered STN: 4 Jun 2004

Last Updated on STN: 4 Jun 2004

\*ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS\*

AB We tested the hypothesis that oxidant-injured cells upregulate thioredoxin, whereas oxidant-stressed, but not injured, cells upregulate interleukin (IL)-8 after injury. We exposed primary human tracheobronchial epithelial cells and transformed human bronchial epithelial cells (BEAS-2B S.6) to 0, 200, 400, or 600 muM H2O2 for 1 h followed by an additional 7 h of incubation. Subsequently, the cells were double-labeled with markers of injury (either Ethidium Homodimer-1 for cellular injury or MitoTracker dye for functional mitochondria) or oxidant stress (5-[and 6]-chloromethyl-2',7'-dichlorodihydrofluorescein diacetate) and antibodies specific for the chemoattractants IL-8 or thioredoxin. We found significant inverse relationships between numbers and stained chemoattractant volumes of IL-8 and thioredoxin-positive cells with increasing H2O2

dose. Cells with mitochondrial injury produced **thioredoxin** but not IL-8, and oxidant-stressed cells were more likely to produce **thioredoxin** than IL-8. Isolated human neutrophils were more likely to colocalize with **thioredoxin**-positive BEAS-2B S.6 cells than **thioredoxin**-negative cells. The H<sub>2</sub>O<sub>2</sub> injury did not induce significant apoptosis in the BEAS-2B S.6 cells as measured by caspase 3 activation. We conclude that oxidant-injured and stressed airway epithelial cells upregulate **thioredoxin**, but produce little IL-8, which may be important in airway epithelial cell-mediated multistep navigation of neutrophils to sites of oxidant injury.

L9 ANSWER 10 OF 37 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2005:524860 BIOSIS

DOCUMENT NUMBER: PREV200510314823

TITLE: **Thioredoxin**-interacting protein controls endothelial cell apoptosis and oxidative stress in hyperglycemia.

AUTHOR(S): Schulze, P. Christian [Reprint Author]; Yoshioka, Jun; Lee, Richard T.

CORPORATE SOURCE: Harvard Univ, Sch Med, Brigham and Womens Hosp, Boston, MA USA

SOURCE: Circulation, (OCT 26 2004) Vol. 110, No. 17, Suppl. S, pp. 260.

Meeting Info.: 77th Scientific Meeting of the American-Heart-Association. New Orleans, LA, USA. November 07 -10, 2004. Amer Heart Assoc. CODEN: CIRCAZ. ISSN: 0009-7322.

DOCUMENT TYPE: Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)

LANGUAGE: English

ENTRY DATE: Entered STN: 1 Dec 2005

Last Updated on STN: 1 Dec 2005

AB Increased oxidative stress plays a central role in the progression of diabetic vascular disease. A major thiol-reducing cellular mechanism is the antioxidative **thioredoxin** system which is regulated by its inhibitor **thioredoxin**-interacting protein (Txnip). In addition to its antioxidative function, **thioredoxin** controls apoptotic cellular pathways by binding and inactivating apoptosis-signaling kinase-1 (ASK-1). We investigated the role of Txnip/**thioredoxin** in hyperglycemia-induced oxidative stress and apoptosis. Incubation of bovine aortic endothelial cells (BAEC) with D-glucose increased reactive oxygen species (ROS) measured by flow cytometry using the redox-sensitive dye DCFDA (186 +/- 10% vs. controls; p<0.001). In addition, **thioredoxin** activity assessed by the insulin-reduction-assay showed a glucose-dependent decrease (-36.8 +/- 23.4%, p<0.05 vs. controls). Hyperglycemia induced robust gene expression of Txnip (>10-fold after 4h at 22.4 mM glucose; p<0.05 vs. controls) and increased protein levels of Txnip by 640% (p<0.05 vs. controls). In contrast, mRNA and protein levels of **thioredoxin** were not altered by glucose. Since Txnip competes with the pro-apoptotic protein ASK-1 for binding to **thioredoxin**, we investigated the effects of Txnip on endothelial apoptosis. Hyperglycemia increased apoptosis in endothelial cells (sub-G1 phase: 3.91 +/- 2.55 vs. 1.48 +/- 0.3 % in controls; p<0.05). Adenoviral overexpression of Txnip increased apoptosis by 4.58 +/- 0.6% vs. 3.01 +/- 0.7% in controls (p<0.001). Increased endothelial apoptosis by Txnip overexpression was confirmed by annexin V staining (p<0.05 vs. controls). Immunoprecipitation revealed reduced ASK-1/**thioredoxin** interaction in cells overexpressing Txnip indicating reduced binding of ASK-1 to **thioredoxin** in the presence of increased cellular levels of Txnip. Intriguingly, gene silencing of Txnip resulted in a reduced endothelial cell apoptosis in hyperglycemia (-49 +/- 12%; p<0.001 vs. scrambled RNAi). Thus, glucose-induced expression of Txnip inhibits the antioxidative

thioredoxin system, promoting oxidative stress and apoptosis in hyperglycemia. Our findings demonstrate a new potential mechanism of hyperglycemia-induced vascular injury.

L9 ANSWER 11 OF 37 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2005:480423 BIOSIS  
DOCUMENT NUMBER: PREV200510267008  
TITLE: An experimental study of gene **expressing** profile in human myeloma cell line RPM18226 induced by thalidomide.  
AUTHOR(S): Zhang, Mei [Reprint Author]; Liu, Ting; Yang, Bingjing; He, Pengcheng; Wang, Mengchang  
CORPORATE SOURCE: Xian Jiaotong Univ, Hosp 1, Xian 710049, Shaanxi, Peoples R China  
SOURCE: Blood, (NOV 16 2004) Vol. 104, No. 11, Part 2, pp. 173B. Meeting Info.: 46th Annual Meeting of the American-Society-of-Hematology. San Diego, CA, USA. December 04 -07, 2004. Amer Soc Hematol. CODEN: BLOOAW. ISSN: 0006-4971.  
DOCUMENT TYPE: Conference; (Meeting)  
Conference; Abstract; (Meeting Abstract)  
LANGUAGE: English  
OTHER SOURCE: GenBank-NM015685; GenBank-AK025983; GenBank-NM000994; GenBank-NM001551; GenBank-NM001033; GenBank-NM001976; GenBank-NM003330; GenBank-NM005167; GenBank-NM005053; GenBank-NM003564; GenBank-NM017432; GenBank-NM001465; GenBank-NM002388; GenBank-BC008861; GenBank-NM001640; GenBank-NM020040; GenBank-NM004341; GenBank-NM003335; GenBank-NM004184  
ENTRY DATE: Entered STN: 16 Nov 2005  
Last Updated on STN: 16 Nov 2005

AB Multiple myeloma (MM) is one of malignant plasmacyte neoplasm in hematopoietic system. Although nearly 70% patients of myeloma response to chemotherapy, but repeated therapies will induce drug resistance soon and lead to refractory or relapsed myelomas. In recent years, thalidomide is used to treat relapsed and refractory myeloma with satisfied effects and overall therapeutic rate with thalidomide is about 60%. Furthermore, the adverse effects of thalidomide is slight without myelosuppression, hepatotoxicity and renal toxicity. So thalidomide is likely to be a prospective antitumor agent. However, the mechanism of antitumor activity of the agent is still not clear. DNA microarray technology has provided us a very useful method to detect simultaneously the **expression** pattern of thousands of genes for investigating the molecular antitumor mechanism of thalidomide. To investigate the genes **expression** profiles of multiple myeloma cell line RPM18226 treated with thalidomide, cDNA microarray were used to detect thousands of gene **expression** in a chip. Two cDNA probes were prepared through reverse transcription from mRNA of RPM18226 cells with or without thalidomide treatment. The probes were labeled with Cy3 and Cy5 fluorescence **dyes** individually, hybridized with cDNA microarray representing 1152 different human genes. Fluorescent intensity were scanned and screened by means of differential analysis between two gene **expression** profiles. After 72 hrs' co-culture of RPM18226 cells and thalidomide in 100  $\mu$ mol/L concentration, the **expression** of 18 genes were up-regulated and 4 genes were down-regulated. The up-regulated genes (GeneBank Accession) included: 1) protein synthesis-related genes: NM\_004184 (WARS), NM\_003335 (UBE1L); 2) immune-related protein: NM\_001465 (FYB), NM\_004341 (CAD), NM\_002388 (MCM3); 3) metabolism related genes: BC008861, NM\_001640 (APEH), NM\_020040 (TUBB4Q), NM\_001033 (RRM1), NM\_001976 (ENO3), NM\_003330 (TXNRDI); 4) cell signals and transducing proteins: NM\_005167 (ARHC), NM\_001465 (FYB); 5) other genes: NM\_017432 (PTOV1), NM\_003564 (TAGLN2), NM\_005053 (RAD23A), NM\_001033 (RRM1), AK025983, NM\_015685 (CLONE24904), NM\_033158 (HYAL2). The down-regulated genes includes: 1) protein synthesis-related genes: NM\_000994 (RPL32); 2).

immune-related proteins: NM\_001551 (IGBP1); 3) other genes: NM\_002983 (SCYA3), NM\_002421 (MMP1). These genes were involved in preprotein synthesis and degradation, cell signal transduction, cytoskeletal movement immune cell metabolism and regulation of anti-oncogene. WARS gene encoding tryptophanyl-tRNA synthetase was up-regulated by thalidomide, while MMP1 gene encoding matrix metalloprotein 1 was down-regulated. They may be related to the inhibition of angiogenesis caused by thalidomide. SCYA3 gene encoding macrophage inflammatory protein-1 alpha was down-regulated by thalidomide, as well as IGBP1 gene which encoding immunoglobulin binding protein I. They may play a role in the inhibition of cell proliferation caused by thalidomide. TUBB4Q gene encoding tubulin beta 4, UBE1L gene encoding ubiquitin-activating enzyme E1-like protein and TXNRDI gene encoding thioredoxin reductase I were up-regulated by thalidomide. They may involve in apoptosis of RPM18226 cells induced by thalidomide. FYB gene encoding Fyn-binding protein was up regulated by thalidomide. The elevated expression of this gene may play a role in the killing of RPM18226 cells by thalidomide.

L9 ANSWER 12 OF 37 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2004449221 EMBASE  
 TITLE: Quantitative proteome analysis in benign thyroid nodular disease using the fluorescent ruthenium II tris(bathophenanthroline disulfonate) stain.  
 AUTHOR: Berger K.; Wissmann D.; Ihling C.; Kalkhof S.; Beck-Sickinger A.; Sinz A.; Paschke R.; Fuhrer D.  
 CORPORATE SOURCE: fued@medizin.uni-leipzig.de  
 SOURCE: Molecular and Cellular Endocrinology, (30 Nov 2004) Vol. 227, No. 1-2, pp. 21-30. .  
 Refs: 14  
 ISSN: 0303-7207 CODEN: MCEND6  
 PUBLISHER IDENT.: S 0303-7207(04)00309-0  
 COUNTRY: Ireland  
 DOCUMENT TYPE: Journal; Article  
 FILE SEGMENT: 003 Endocrinology  
 022 Human Genetics  
 029 Clinical Biochemistry  
 LANGUAGE: English  
 SUMMARY LANGUAGE: English  
 ENTRY DATE: Entered STN: 4 Nov 2004  
 Last Updated on STN: 4 Nov 2004

AB Thyroid tumorigenesis involves qualitative and quantitative changes in protein expression, which can be comprehensively studied by proteome analysis. However, one of the technical bottlenecks of proteomics remains a reliable, sensitive and inexpensive method for quantification of differentially expressed proteins. This is due to the limited linear range of most available protein stains, i.e. silver and Coomassie blue, and high costs of commercially available fluorescent stains. In this paper we describe our experience with a lab-made ruthenium based fluorescent stain (ruthenium II tris(bathophenanthroline disulfonate) (RuBPs)) to perform proteome analysis of nodular thyroid disease. We first compared the properties of RuBPs with two highly sensitive protein stains: (1) silver staining and (2) the commercially available fluorescent dye Sypro Ruby. We show that in addition to its highly sensitive staining capabilities similar to Sypro Ruby and silver (2 ng), RuBPs offers several advantages such as a broad dynamic range (similar to Sypro Ruby and 500 times broader than the dynamic range of silver stain), low costs ((euro) 0.03 per gel) and excellent compatibility with mass spectrometry. We then applied the inexpensive RuBPs stain to 2D gels (pH 4-7) of four benign thyroid nodules and normal thyroid tissue. We were able to detect approx. 1800 protein spots/gel in our thyroid samples. Quantitative changes in protein expression levels of at least 20-42 proteins were noted in the benign nodules compared with the normal thyroid tissue of the same

patient. Differentially **expressed** spots were further characterised by nano-LC-FTICR and MALDI-TOF mass spectrometry. In summary we demonstrate, that the novel fluorescent ruthenium II tris(bathophenanthroline disulfonate) stain is a highly sensitive, reliable and inexpensive tool for quantitative proteome analysis in thyroid nodular disease. .COPYRG. 2004 Elsevier Ireland Ltd. All rights reserved.

L9 ANSWER 13 OF 37 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2003:591440 HCAPLUS

DOCUMENT NUMBER: 139:148006

TITLE: Comparative gene profiling between undifferentiated and well-differentiated nasopharyngeal carcinoma (NPC) cells for NPC diagnosis and treatment

INVENTOR(S): Ng, Aylwin; Tang, Jing P.; Hui, Kam M.; Goh, Christopher H. K.

PATENT ASSIGNEE(S): Biotech Research Ventures Pte Limited, Singapore; Cripps, Joanna E.

SOURCE: PCT Int. Appl., 61 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003062826	A2	20030731	WO 2003-GB329	20030123
WO 2003062826	A3	20031016		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CN 1643377	A	20050720	CN 2003-806030	20030123
PRIORITY APPLN. INFO.:			GB 2002-1498	A 20020123

AB The invention is concerned with the detection and treatment of nasopharyngeal carcinoma (NPC) based on differential gene **expression** in these cells. Specifically, the invention provides details of differentially **expressed** genes in NPC which serve to detect the presence or risk of the disease and its clin. type. A set of genes are identified by DNA microarray using comparative gene profiling between undifferentiated and well-differentiated nasopharyngeal carcinoma (NPC) cells (CNE-2 or HK1 resp.) to be associated with NPC, in particular, two imprinted genes H19 (no protein product, function unknown) and CDKN1C (cyclin-dependent kinase inhibitor 1C) located on chromosome 11p15. It is also shown H19 gene is highly **expressed** in CNE-2 cells and hypermethylation of its CpG promoter region is detected in the HK1 cells. Furthermore, hypomethylation of the CpG dinucleotides within H19 promoter region is correlated with the restoration of its mRNA **expression** in HK1 cells. The invention also provides methods of treating NPC in association with chemo or radiotherapy.

L9 ANSWER 14 OF 37 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2003:334399 HCAPLUS

DOCUMENT NUMBER: 138:352748

TITLE: WT1 protein-derived immunogenic peptides for immunotherapy of leukemia and cancer

INVENTOR(S): Gaiger, Alexander; Cheever, Martin A.; McNeill,

PATENT ASSIGNEE(S): Patricia D.; Smithgall, Molly; Moulton, Gus; Vedvick, Thomas S.; Sleath, Paul R.  
 SOURCE: USA  
 U.S. Pat. Appl. Publ., 161 pp., Cont.-in-part of U. S. Ser. No. 685,830.  
 CODEN: USXXCO  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 FAMILY ACC. NUM. COUNT: 11  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003082196	A1	20030501	US 2001-785019	20010215
US 2003072767	A1	20030417	US 2001-938864	20010824
CA 2425072	AA	20020411	CA 2001-2425072	20011003
WO 2002028414	A1	20020411	WO 2001-US31139	20011003
WO 2002028414	B1	20020718		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2001096608	A5	20020415	AU 2001-96608	20011003
EP 1328287	A1	20030723	EP 2001-977493	20011003
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
JP 2004510425	T2	20040408	JP 2002-532238	20011003
CN 1505526	A	20040616	CN 2001-819114	20011003
US 2003095971	A1	20030522	US 2001-2603	20011030
US 2003039635	A1	20030227	US 2002-125635	20020416
US 2003198622	A1	20031023	US 2002-195835	20020712
US 2003235557	A1	20031225	US 2002-244830	20020916
US 2003215458	A1	20031120	US 2002-286333	20021030
US 2004126362	A1	20040701	US 2003-648780	20030826
AU 2003257511	A1	20031120	AU 2003-257511	20031023

PRIORITY APPLN. INFO.:  
 US 2000-685830 A2 20001009  
 US 1998-164223 A2 19980930  
 US 1999-276484 A2 19990325  
 AU 1999-64078 A3 19990930  
 US 2000-684361 A2 20001006  
 US 2001-785019 A2 20010215  
 US 2001-938864 A 20010824  
 WO 2001-US31139 W 20011003  
 US 2001-2603 A2 20011030  
 US 2002-125635 A2 20020416  
 US 2002-195835 A2 20020712  
 US 2002-244830 A2 20020916

AB Compns. and methods for the therapy of malignant diseases, such as leukemia and cancer, are disclosed. The compns. comprise one or more of a WT1 polynucleotide, a WT1 polypeptide, an antigen-presenting cell presenting a WT1 polypeptide, an antibody that specifically binds to a WT1 polypeptide; or a T cell that specifically reacts with a WT1 polypeptide. Such compns. may be used, for example, for the prevention and treatment of metastatic diseases.

L9 ANSWER 15 OF 37 MEDLINE on STN  
 ACCESSION NUMBER: 2003126656 MEDLINE  
 DOCUMENT NUMBER: PubMed ID: 12514184  
 TITLE: Catalytic mechanism of thiol peroxidase from Escherichia



coli. Sulfenic acid formation and overoxidation of essential CYS61.

AUTHOR: Baker Laura M S; Poole Leslie B

CORPORATE SOURCE: Department of Biochemistry, Wake Forest University School of Medicine, Winston-Salem, North Carolina 27157, USA.

CONTRACT NUMBER: R01 GM50389 (NIGMS)

SOURCE: The Journal of biological chemistry, (2003 Mar 14) Vol. 278, No. 11, pp. 9203-11. Electronic Publication: 2003-01-03.

Journal code: 2985121R. ISSN: 0021-9258.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200305

ENTRY DATE: Entered STN: 20030319  
Last Updated on STN: 20030515  
Entered Medline: 20030514

AB Escherichia coli thiol peroxidase (Tpx, p20, scavengase) is part of an oxidative stress defense system that uses reducing equivalents from thioredoxin (Trx1) and thioredoxin reductase to reduce alkyl hydroperoxides. Tpx contains three Cys residues, Cys(95), Cys(82), and Cys(61), and the latter residue aligns with the N-terminal active site Cys of other peroxidases in the peroxiredoxin family. To identify the catalytically important Cys, we have cloned and purified Tpx and four mutants (C61S, C82S, C95S, and C82S,C95S). In rapid reaction kinetic experiments measuring steady-state turnover, C61S is inactive, C95S retains partial activity, and the C82S mutation only slightly affects reaction rates. Furthermore, a sulfenic acid intermediate at Cys(61) generated by cumene hydroperoxide (CHP) treatment was detected in UV-visible spectra of 4-nitrobenzo-2-oxa-1,3-diazole-labeled C82S,C95S, confirming the identity of Cys(61) as the peroxidatic center. In stopped-flow kinetic studies, Tpx and Trx1 form a Michaelis complex during turnover with a catalytic efficiency of  $3.0 \times 10^6 \text{ m}^{-1} \text{ s}^{-1}$ , and the low  $K_m$  (9.0 microm) of Tpx for CHP demonstrates substrate specificity toward alkyl hydroperoxides over  $\text{H}_2\text{O}_2$  ( $K_m > 1.7 \text{ mM}$ ). Rapid inactivation of Tpx due to Cys(61) overoxidation is observed during turnover with CHP and a lipid hydroperoxide, 15-hydroperoxyeicosatetraenoic acid, but not  $\text{H}_2\text{O}_2$ . Unlike most other 2-Cys peroxiredoxins, which operate by an intersubunit disulfide mechanism, Tpx contains a redox-active intrasubunit disulfide bond yet is homodimeric in solution.

L9 ANSWER 16 OF 37 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2004:25886 BIOSIS

DOCUMENT NUMBER: PREV200400024278

TITLE: PROBIOTICS CHANGE E. COLI-INDUCED GENE EXPRESSION PROFILES IN CACO-2 CELLS.

AUTHOR(S): Panigrahi, Pinaki [Reprint Author]; Braileanu, Gheorghe [Reprint Author]

CORPORATE SOURCE: Baltimore, MD, USA

SOURCE: Digestive Disease Week Abstracts and Itinerary Planner, (2003) Vol. 2003, pp. Abstract No. T1082. e-file.  
Meeting Info.: Digestive Disease 2003. FL, Orlando, USA. May 17-22, 2003. American Association for the Study of Liver Diseases; American Gastroenterological Association; American Society for Gastrointestinal Endoscopy; Society for Surgery of the Alimentary Tract.

DOCUMENT TYPE: Conference; (Meeting)  
Conference; Abstract; (Meeting Abstract)  
Conference; (Meeting Poster)

LANGUAGE: English

ENTRY DATE: Entered STN: 31 Dec 2003

Last Updated on STN: 31 Dec 2003

AB Background: Lactobacillus group of organisms (probiotics) are known for their protective role in infectious and inflammatory conditions of the intestine. In our previous studies, Lactobacillus plantarum (Lp) has been shown to block E. coli-induced tissue injury in a weanling rabbit ileal loop model via modulation of inflammatory response. We have further reported the **expression** of specific genes including enterocyte differentiation factor, aquaporin 3, nucleoporin, oxidoreductase and thioredoxin reductase 2 by Lp in Caco-2 cells. Aim: In this study, DNA microarray technique was used to examine the induction of genes after infection with (i) E. coli, (ii) Lp, and (iii) the combination (E. coli+Lp in co-culture) in cultured Caco-2 cells.. Methods: Differentiated Caco2 cells were treated for 2 hr with E. coli (109 CFU/ml), Lp (109 CFU/ml) or the combination. Total RNA was extracted using the Trizol method. A 3DNA submicro labeling kit for 2 step protocol (Genisphere) was used for hybridization in a glass microarray with 19 000 human ESTs/genes in duplicate (University of Toronto). Samples (control and three experimental conditions) were labeled with Alexa 488, 546 594 and 647 dyes, scanned with 4 different lasers, and analyzed (including hierarchical cluster analysis) using the ImaGene 4.2. and GeneSight 3 (Biodiscovery Inc.) softwares. Results: A set of 323, 347 and 262 genes/ESTs were induced by E. coli, Lp and the combination respectively. Interestingly, of these genes, while E. coli and Lp stimulated 203 and 221 genes respectively, the co-infection resulted in only the stimulation of 160 genes. E. coli and LP suppressed 120, and 126 genes respectively with only 102 genes suppressed during mixed infection. Of the 323 and 262 genes **expressed** during E. coli and mixed infection, 170 gene were found to be common, and there were 153 and 92 unique genes in these two conditions. Further analysis of these genes/ESTs are currently underway in our laboratory. Conclusions: Although **expression** of different sets of genes was expected by specific infections in this experiment, it was intriguing to note that addition of Lp changed (reduced) the primary genes induced by E. coli alone. The phenomenon of 'reversal' of activation and repression (by E. coli) of gene sets by Lp as observed in this study may point to the inherent ability of probiotics in affecting eukaryotic genes and thereby modulating the infective and inflammatory events induced by normal flora and pathogenic bacteria in the intestine..

L9 ANSWER 17 OF 37 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2002:267906 BIOSIS  
DOCUMENT NUMBER: PREV200200267906  
TITLE: Molecular characterization of a novel protein disulfide isomerase in carrot.  
AUTHOR(S): Xu, Zheng-Jun; Ueda, Kenji; Masuda, Kiyoshi; Ono, Michiyuki; Inoue, Masayasu [Reprint author]  
CORPORATE SOURCE: Biotechnology Institute, Akita Prefectural University, Ohgata, Akita, 010-0444, Japan  
ino@agri.akita-pu.ac.jp  
SOURCE: Gene (Amsterdam), (6 February, 2002) Vol. 284, No. 1-2, pp. 225-231. print.  
CODEN: GENED6. ISSN: 0378-1119.  
DOCUMENT TYPE: Article  
LANGUAGE: English  
OTHER SOURCE: DDBJ-D85891  
ENTRY DATE: Entered STN: 1 May 2002  
Last Updated on STN: 1 May 2002

AB A protein disulfide isomerase (PDI) coding sequence was cloned from a cDNA library derived from carrot (Daucus carota L.) somatic embryos. The cDNA is 2060 bp in length and encodes for a protein of 581 amino acids and molecular weight of 64.4 kDa. Primary structure analysis of the deduced protein revealed two thioredoxin-like active sites and an endoplasmic reticulum-retention signal at its C-terminus,

which is also found in PDIs in plants and animals. Although between the carrot protein and other plant PDIs there is only about 30% identity, the active site regions are almost identical. The corresponding mRNA was found in varying amounts, in all tissues investigated. A recombinant protein expressed from the carrot cDNA clone effectively catalyzed both glutathione-insulin transhydrogenation and the oxidative renaturation of denatured RNase A. These results suggest that the protein coded for by the carrot gene is a novel member of the PDI family in plants. We therefore designated this novel carrot gene PDIL1. The protein expressed by the PDIL1 cDNA sequence had a highly acidic stretch at its N-terminal region (no such domain exists in known plant PDIs), and was located far from known plant PDIs on a maximum likelihood tree. The PDIL1 gene, together with closely-related genes identified in Arabidopsis and tomato, was suggested to belong to a novel subfamily of PDIs.

L9 ANSWER 18 OF 37 MEDLINE on STN  
 ACCESSION NUMBER: 2002094790 MEDLINE  
 DOCUMENT NUMBER: PubMed ID: 11792180  
 TITLE: Poly[Lys-(AEDTP)]: a cationic polymer that allows dissociation of pDNA/cationic polymer complexes in a reductive medium and enhances polyfection.  
 AUTHOR: Pichon Chantal; LeCam Eric; Guerin Brigitte; Coulaud Dominique; Delain Etienne; Midoux Patrick  
 CORPORATE SOURCE: Centre de Biophysique Moleculaire, CNRS UPR 4301, rue Charles Sadron, F-45071 Orleans Cedex 02, France.  
 SOURCE: Bioconjugate chemistry, (2002 Jan-Feb) Vol. 13, No. 1, pp. 76-82.  
 Journal code: 9010319. ISSN: 1043-1802.  
 PUB. COUNTRY: United States  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 200204  
 ENTRY DATE: Entered STN: 20020205  
 Last Updated on STN: 20020406  
 Entered Medline: 20020405

AB Polyplexes of high stability resulting from the condensation of a plasmid DNA by a cationic polymer are widely used to develop polymer-based gene delivery systems. However, the plasmid must be released from its vector once inside the cells for an efficient expression of the exogenous gene in the cell nucleus. We have designed a disulfide-containing cationic polymer termed poly[Lys-(AEDTP)] which allowed for the formation of polyplexes and the release of the plasmid in a reductive medium. The amino groups of polylysine were substituted with 3-(2-aminoethylthio)propionyl residues in order to have each amino group of poly[Lys-(AEDTP)] interacting with a phosphate DNA linked to the polymer backbone via a disulfide bond. As evidenced by agarose gel electrophoresis and ethidium bromide/pDNA fluorescence restoration, poly[Lys-(AEDTP)] polyplexes were decondensed and the plasmid released upon treatment with either dithiothreitol, glutathione in the presence of glutathione reductase, or the thioredoxin reductase. Electron microscopy showed that polyplexes exhibiting spherical particles of a mean size at about 100 nm were decondensed in the presence of glutathione and exhibited filamentous aggregates. Finally, we found that the transfection of 293T7 and HepG2 cells was 10- and 50-fold more efficient with poly[Lys-(AEDTP)] polyplexes, respectively, than with poly[Lys] polyplexes. These results indicate that disulfide-containing cationic polymers must be borne in mind for developing polymer-base gene delivery systems.

L9 ANSWER 19 OF 37 NTIS COPYRIGHT 2006 NTIS on STN  
 ACCESSION NUMBER: 2001(16):04701  
 NTIS ORDER NUMBER: ADA388146/XAB

TITLE: Biomimetic Sensor for Pathogenic Bacteria. Final rept 1  
 May-Sep 30 2000.  
 AUTHOR: Walsh, M. K.; De Wald, D. B.; Weimer, B. C.  
 CORPORATE SOURCE: Utah State Univ., Logan. Dept. of Nutrition and Food  
 Science. (004748080 407359)  
 NUMBER OF REPORT: ADA388146/XAB; AFRL; SR-BL-TR-01-0190  
 17p; 5 Mar 2001  
 NUMBER OF CONTRACT: F49620-00-1-0289  
 2312  
 DX  
 CONTROLLED TERM: Report  
 COUNTRY: United States  
 LANGUAGE: English  
 AVAILABILITY: Product reproduced from digital image. Order this  
 product from NTIS by: phone at 1-800-553-NTIS (U.S.  
 customers); (703)605-6000 (other countries); fax at  
 (703)605-6900; and email at orders@ntis.gov. NTIS is  
 located at 5285 Port Royal Road, Springfield, VA,  
 22161, USA.  
 NTIS Prices: PC A03/MF A01  
 OTHER SOURCE: GRA&I0115

AB This study investigated the assembly of a biomimetic sensor containing  
 an osmotic receptor protein. The first objective of this research  
 included the assembly and immobilization of fluorescently tagged  
 liposomes. The second objective involved the **expression** and  
 purification of an osmotic sensitive protein (MscL) and the  
 incorporation of this protein into the liposome membrane. Liposomes (2  
 micron diameter) containing fluorescein labeled phospholipids and  
 biotinylated phosphatidyl ethanolamine in the membranes and internalized  
 soluble sulforhodamine were assembled. Liposomes were characterized with  
 respect to composition, size, and shelf-life using confocal microscopy.  
 Avidin was covalently attached to a glass surface for the immobilization  
 of the biotinylated liposomes. Immobilization of fluorescent liposomes  
 was confirmed with confocal microscopy. The liposomes contain a  
 green/yellow lipid bilayer and a red interior. The **cloning** of  
**recombinant** MscL into an Escherichia coli **expression**  
 system yielded an MscL-thioredoxin fusion protein that was  
 tagged with a blue fluorescent **dye** and incorporated into the  
 membrane of the liposomes. The functionality of the proteosome was  
 observed by the release of the water soluble sulforhodamine in the  
 presence of high salt, 3 M, concentrations.

L9 ANSWER 20 OF 37 MEDLINE on STN  
 ACCESSION NUMBER: 2001518508 MEDLINE  
 DOCUMENT NUMBER: PubMed ID: 11565801  
 TITLE: Proteomic characterization of early-stage differentiation  
 of mouse embryonic stem cells into neural cells induced by  
 all-trans retinoic acid in vitro.  
 AUTHOR: Guo X; Ying W; Wan J; Hu Z; Qian X; Zhang H; He F  
 CORPORATE SOURCE: Department of Genomics and Proteomics, Beijing Institute of  
 Radiation Medicine, China.  
 SOURCE: Electrophoresis, (2001 Aug) Vol. 22, No. 14, pp. 3067-75.  
 Journal code: 8204476. ISSN: 0173-0835.  
 PUB. COUNTRY: Germany; Germany, Federal Republic of  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 200204  
 ENTRY DATE: Entered STN: 20010924  
 Last Updated on STN: 20020409  
 Entered Medline: 20020408

AB Embryonic stem (ES) cells are totipotent stem cells, which can  
 differentiate into various kinds of cell types, including neurons. They  
 are widely used as a model system for investigating mechanisms of

differentiation events during early mouse development. In this study, proteomic techniques were used to approach the protein profile associated with the early-stage differentiation of ES cells into neuronal cells induced by all-trans retinoic acid (ATRA) in vitro. In comparison of the protein profile of parent ES cells with that of ES-derived neural-committed cells, which was induced by ATRA for four days, 24 differentially displayed protein spots were selected from two-dimensional electrophoresis (2-DE) gels for further protein identification by peptide mass fingerprinting (PMF). Nine proteins were known to being involved in the process of neural differentiation and/or neural survival. Of those, alpha-3/alpha-7 tubulin and vimentin were down-regulated, while cytokeratin 8, cytokeratin 18, G1/S-special cyclin D2, follistatin-related protein, NEL protein, platelet-activating factor acetylhydrolase IB alpha-subunit, and thioredoxin peroxidase 2 were upregulated during differentiation of ES cells to neural cells. Additionally, other 12 protein (five upregulated and seven downregulated) spots associated with ES cell differentiation into neuronal cells were not matched to known proteins so far, implicating that they might be novel proteins. The results above indicated that the molecular mechanisms of differentiation of ES cells to neural cells in vitro might be similar to those of other neural systems in vitro and identified that proteomic analysis is an effective strategy to comprehensively unravel the regulatory network of differentiation.

L9 ANSWER 21 OF 37 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2001:98372 HCAPLUS

DOCUMENT NUMBER: 134:232542

TITLE: Genome sequence of enterohaemorrhagic Escherichia coli O157:H7

AUTHOR(S): Perna, Nicole T.; Plunkett, Guy, III; Burland, Valerie; Mau, Bob; Glasner, Jeremy D.; Rose, Debra J.; Mayhew, George F.; Evans, Peter S.; Gregor, Jason; Kirkpatrick, Heather A.; Posfai, Gyorgy; Hackett, Jeremiah; Klink, Sara; Boutin, Adam; Shao, Ying; Miller, Leslie; Grotbeck, Erik J.; Davis, N. Wayne; Lim, Alex; Dimalanta, Eileen T.; Potamousis, Konstantinos D.; Apodaca, Jennifer; Anantharaman, Thomas S.; Lin, Jieyi; Yen, Glaex; Schwartz, David C.; Welch, Rodney A.; Blattner, Frederick R.

CORPORATE SOURCE: Genome Center of Wisconsin, Department of Animal Health and Biomedical Sciences, Laboratory of Genetics, Department of Chemistry, Department of Biostatistics, and Department of Medical Microbiology and Immunology, University of Wisconsin, Madison, WI, 53706, USA

SOURCE: Nature (London) (2001), 409(6819), 529-533

CODEN: NATUAS; ISSN: 0028-0836

PUBLISHER: Nature Publishing Group

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The bacterium Escherichia coli O157:H7 is a worldwide threat to public health and has been implicated in many outbreaks of hemorrhagic colitis, some of which included fatalities caused by hemolytic uremic syndrome. Close to 75,000 cases of O157:H7 infection are now estimated to occur annually in the United States. The severity of disease, the lack of effective treatment and the potential for large-scale outbreaks from contaminated food supplies have propelled intensive research on the pathogenesis and detection of E. coli O157:H7. The genome of E. coli O157:H7 was sequenced to identify candidate genes responsible for pathogenesis, to develop better methods of strain detection and to advance our understanding of the evolution of E. coli, through comparison with the genome of the non-pathogenic laboratory strain E. coli K-12. Lateral gene transfer found to be far more extensive than previously anticipated. In fact, 1387 new genes encoded in strain-specific clusters of diverse sizes were found in

0157:H7. These include candidate virulence factors, alternative metabolic capacities, several prophages, and other new functions - all of which could be targets for surveillance.

REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 22 OF 37 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2001:209011 HCAPLUS

DOCUMENT NUMBER: 137:196299

TITLE: Genome sequence of enterohaemorrhagic Escherichia coli 0157:H7. [Erratum to document cited in CA134:232542]

AUTHOR(S): Perna, Nicole T.; Plunkett, Guy, III; Burtand, Valerie; Mau, Bob; Glasner, Jeremy D.; Rose, Debra J.; Mayhew, George F.; Evans, Peter S.; Gregor, Jason; Kirkpatrick, Heather A.; Postal, Gyorgy; Hackett, Jeremiah; Klink, Sara; Boutin, Adam; Shao, Ying; Miller, Leslie; Grotbeck, Erik J.; Davis, N. Wayne; Lim, Alex; Dimalanta, Eileen T.; Potamouisis, Konstantinos D.; Apodaca, Jennifer; Anantharaman, Thomas S.; Lin, Jleyl; Yen, Galex; Schwartz, Dvauid C.; Welch, Rodney A.; Blatner, Frederick R.

CORPORATE SOURCE: Genome Center of Wisconsin, Department of Animal Health and Biomedical Sciences, Laboratory of Genetics, Department of Chemistry, Department of Biostatistics, and Department of Medical Microbiology and Immunology, University of Wisconsin, Madison, WI, 53706, USA

SOURCE: Nature (London, United Kingdom) (2001), 410(6825), 240  
CODEN: NATUAS; ISSN: 0028-0836

PUBLISHER: Nature Publishing Group

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The correct GenBank accession number for the annotated sequence is AE005174.

L9 ANSWER 23 OF 37 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2001:209816 BIOSIS

DOCUMENT NUMBER: PREV200100209816

TITLE: Fluorescently labeled model DNA sequences for exonucleolytic sequencing.

AUTHOR(S): Foeldes-Papp, Zeno [Reprint author]; Angerer, Bernhard; Thyberg, Per; Hinz, Michael; Wennmalm, Stefan; Ankenbauer, Waltraud; Seliger, Hartmut; Holmgren, Arne; Rigler, Rudolf

CORPORATE SOURCE: Clinical Immunology and Jean Dausset Laboratory, Graz University M.S. and Hospital, Auenbrugger Platz 8, A-8036, Graz, Austria  
Zeno.Foldes-Papp@kfunigraz.ac.at

SOURCE: Journal of Biotechnology, (13 April, 2001) Vol. 86, No. 3, pp. 203-224. print.  
CODEN: JBITD4. ISSN: 0168-1656.

DOCUMENT TYPE: Article

LANGUAGE: English

ENTRY DATE: Entered STN: 2 May 2001

Last Updated on STN: 18 Feb 2002

AB We describe here the enzyme-catalyzed, low-density labeling of DNAs with fluorescent dyes. Firstly, for 'natural' template DNAs, dNTPs were partially substituted in the labeling reactions by the respective fluorophore-bearing analogs. The DNAs were labeled by PCR using Taq DNA polymerase. The covalent incorporation of dye-dNTPs decreased in the following order: rhodamine-green-5-dUTP (Molecular Probes, the Netherlands), tetramethylrhodamine-4-dUTP (FluoroRed, Amersham Pharmacia Biotech), Cy5-dCTP (Amersham Pharmacia Biotech). Exonucleolytic degradation by the 3' fwdarw 5' exonuclease activity of T7 DNA polymerase (wild type) in the presence of excess reduced thioredoxin

proceeded to complete breakdown of the labeled DNAs. The catalytic cleavage constants determined by fluorescence correlation spectroscopy were between 0.5 and 1.5 s<sup>-1</sup> at 16°C, normalized for the covalently incorporated dye-nucleotides. Secondly, rhodamine-green-X-dUTP (Roche Diagnostics), tetramethylrhodamine-6-dUTP (Roche Diagnostics), and Cy5-dCTP were covalently incorporated into the antisense strand of 'synthetic' 218-b DNA template constructs (master sequences) at well defined positions, starting from the primer binding site, by total substitution for the naturally occurring dNTPs. The 218-b DNA constructs were labeled by PCR with a thermostable 3' exonuclease deficient mutant of the Tgo DNA polymerase which we have selected. The advantage of the special, synthetic DNA constructs as compared to natural DNAs lies in the possibility of obtaining tailor-made nucleic acids, optimized for testing the performance of exonucleolytic sequencing. The number of incorporated fluorescent nucleotides determined by complete exonucleolytic degradation and fluorescence correlation spectroscopy were six out of six possible incorporations for rhodamine-green-X-dUTP and tetramethylrhodamine-6-dUTP, respectively. Their covalent and base-specific incorporations were confirmed by the novel analysis methodology of re-sequencing (i.e. mobility-shift gel electrophoresis, reversion-PCR and re-sequencing) first developed in the paper Foldes-Papp et al. (2001) and in this paper. This methodology was then used by other groups within the whole sequencing project.

L9 ANSWER 24 OF 37 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2001:184271 HCAPLUS

DOCUMENT NUMBER: 134:217892

TITLE: Complete genome sequence of enterohemorrhagic *Escherichia coli* O157:H7 and genomic comparison with a laboratory strain K-12

AUTHOR(S): Hayashi, Tetsuya; Makino, Kozo; Ohnishi, Makoto; Kurokawa, Ken; Ishii, Kazuo; Yokoyama, Katsushi; Han, Chang-Gyun; Ohtsubo, Eiichi; Nakayama, Keisuke; Murata, Takahiro; Tanaka, Masashi; Tobe, Toru; Iida, Tetsuya; Takami, Hideto; Honda, Takeshi; Sasakawa, Chihiro; Ogasawara, Naotake; Yasunaga, Teruo; Kuhara, Satoru; Shiba, Tadayoshi; Hattori, Masahira; Shinagawa, Hideo

CORPORATE SOURCE: Department of Microbiology, Miyazaki Medical College, Miyazaki, 899-1692, Japan

SOURCE: DNA Research (2001), 8(1), 11-22

CODEN: DARSE8; ISSN: 1340-2838

PUBLISHER: Universal Academy Press

DOCUMENT TYPE: Journal

LANGUAGE: English

AB *Escherichia coli* O157:H7 is a major food-borne infectious pathogen that causes diarrhea, hemorrhagic colitis, and hemolytic uremic syndrome. The complete chromosome sequence of an O157:H7 strain isolated from the Sakai outbreak is reported, and the results compared with the genome of a benign laboratory strain, K-12 MG1655. The chromosome is 5.5 Mb in size, 859 Kb

larger

than that of K-12. A 4.1-Mb sequence highly conserved between the two strains is identified, which may represent the fundamental backbone of the *E. coli* chromosome. The remaining 1.4-Mb sequence comprises of O157:H7-specific sequences, most of which are horizontally transferred foreign DNAs. The predominant roles of bacteriophages in the emergence of O157:H7 is evident by the presence of 24 prophages and prophage-like elements that occupy more than half of the O157:H7-specific sequences. The O157:H7 chromosome encodes 1632 proteins and 20 tRNAs that are not present in K-12. Among these, at least 131 proteins are assumed to have virulence-related functions. Genome-wide codon usage analysis suggested that the O157:H7-specific tRNAs are involved in the efficient expression of the strain-specific genes. A complete set of the genes specific to O157:H7 presented here sheds new insight into the

pathogenicity and the physiolo. of O157:H7, and will open a way to fully understand the mol. mechanisms underlying the O157:H7 infection.

REFERENCE COUNT: 62 THERE ARE 62 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 25 OF 37 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2000:666972 HCAPLUS

DOCUMENT NUMBER: 133:219795

TITLE: Proteins for diagnosis and treatment of breast cancer

INVENTOR(S): Amess, Bob; Townsend, Robert Reid; Parekh, Rajesh  
Bhikhu; Waterfield, Michael Derek; O'Hare, Michael  
John

PATENT ASSIGNEE(S): Oxford Glycosciences (UK) Ltd., UK

SOURCE: PCT Int. Appl., 86 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000055628	A1	20000921	WO 2000-GB908	20000313
W:	AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW:	GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG			
EP 1159618	A1	20011205	EP 2000-909494	20000313
R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO			

PRIORITY APPLN. INFO.: GB 1999-5817 A 19990312  
WO 2000-GB908 W 20000313

AB The present invention provides methods and compns. for screening, diagnosis and prognosis of breast cancer, for monitoring the effectiveness of breast cancer treatment, and for drug development. Breast Cancer-Associated Features (BFs), detectable by two-dimensional electrophoresis of breast tissue, are described. The invention further provides Breast Cancer-Associated Protein Isoforms (BPIs) detectable in breast tissue, preps. comprising isolated BPIs, antibodies immunospecific for BPIs, and kits comprising the aforesaid. Luminal and myoepithelial cells were purified by immunomagnetic methods from 10 sets of matched normal and cancer breast cell tissue. Two-dimensional electrophoresis was used to sep. the proteins. High resolution detection of protein features using fluorescent dyes, coupled to advanced software to identify differentially expressed features, high through-put mass spectrometry and bioinformatics was also applied. This has allowed the identification of large sets of proteins which are differentially expressed between the luminal and myoepithelial human breast cell proteomes.

REFERENCE COUNT: 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 26 OF 37 MEDLINE on STN

ACCESSION NUMBER: 2000290849 MEDLINE

DOCUMENT NUMBER: PubMed ID: 10828978

TITLE: AhpF can be dissected into two functional units: tandem repeats of two thioredoxin-like folds in the N-terminus mediate electron transfer from the thioredoxin reductase-like C-terminus to AhpC.



AUTHOR: Poole L B; Godzik A; Nayeem A; Schmitt J D  
 CORPORATE SOURCE: Department of Biochemistry, Wake Forest University School of Medicine, Winston-Salem, North Carolina 27157, USA..  
 lbpoole@wfubmc.edu  
 CONTRACT NUMBER: GM50389 (NIGMS)  
 GM60049 (NIGMS)  
 SOURCE: Biochemistry, (2000 Jun 6) Vol. 39, No. 22, pp. 6602-15.  
 Journal code: 0370623. ISSN: 0006-2960.  
 PUB. COUNTRY: United States  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 200007  
 ENTRY DATE: Entered STN: 20000720  
 Last Updated on STN: 20021210  
 Entered Medline: 20000710

AB AhpF, the flavin-containing component of the Salmonella typhimurium alkyl hydroperoxide reductase system, catalyzes the NADH-dependent reduction of an active-site disulfide bond in the other component, AhpC, which in turn reduces hydroperoxide substrates. The amino acid sequence of the C-terminus of AhpF is 35% identical to that of **thioredoxin** reductase (TrR) from Escherichia coli. AhpF contains an additional 200-residue N-terminal domain possessing a second redox-active disulfide center also required for AhpC reduction. Our studies indicate that this N-terminus contains a tandem repeat of two **thioredoxin** (Tr)-like folds, the second of which contains the disulfide redox center. Structural and catalytic properties of independently **expressed** fragments of AhpF corresponding to the TrR-like C-terminus (F[208-521]) and the 2Tr-like N-terminal domain (F[1-202]) have been addressed. Enzymatic assays, reductive titrations, and circular dichroism studies of the fragments indicate that each folds properly and retains many functional properties. Electron transfer between F[208-521] and F[1-202] is, however, relatively slow ( $4 \times 10^4$  M<sup>-1</sup> s<sup>-1</sup>) at 25 degrees C) and nonsaturable up to 100 microm F[1-202]. TrR is nearly as efficient at F[1-202] reduction as is F[208-521], although neither the latter fragment, nor intact AhpF, can reduce Tr. An engineered mutant AhpC substrate with a fluorophore attached via a disulfide bond has been used to demonstrate that only F[1-202], and not F[208-521], is capable of electron transfer to AhpC, thereby establishing the direct role this N-terminal domain plays in mediating electron transfer between the TrR-like part of AhpF and AhpC.

L9 ANSWER 27 OF 37 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1999:747450 HCAPLUS  
 DOCUMENT NUMBER: 132:1816  
 TITLE: A protein-labeling compound, and a method for labeling protein using this compound  
 INVENTOR(S): Yanagawa, Hiroshi; Nemoto, Naoto; Miyamoto, Etsuko  
 PATENT ASSIGNEE(S): Mitsubishi Chemical Industries Ltd., Japan  
 SOURCE: Jpn. Kokai Tokkyo Koho, 9 pp.  
 CODEN: JKXXAF  
 DOCUMENT TYPE: Patent  
 LANGUAGE: Japanese  
 FAMILY ACC. NUM. COUNT: 1  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 11322781	A2	19991124	JP 1998-133170	19980515
US 6228994	B1	20010508	US 1998-190276	19981113
US 2001039011	A1	20011108	US 2000-737751	20001218
US 2001007751	A1	20010712	US 2001-794128	20010228
PRIORITY APPLN. INFO.:			JP 1998-133170	A 19980515
			US 1998-190276	A3 19981113

AB A protein-labeling compound is chemical synthesized from puromycin or puromycin

analog and a functional compound by forming a chemical bond between them. This protein-labeling compound is made of a labeling part consisting of a labeling chemical and an acceptor part consisting of a chemical capable of binding to C-terminus of a protein. A labeling of a protein is carried out by adding the protein-labeling compound at the final concentration of 10-0.01 $\mu$ M to the translation system in cell-free extract of prokaryotic or eukaryotic cell, or to living cells. A labeling part chemical can be one of various functional compds. such as a fluorescent dye and a radioactive substance. The protein-labeling compound is useful in detecting or identifying the protein **expressed** in various translation systems of cell-free extract or living cells. The identification of the corresponding proteins is the most important task for the functional anal. of the genes which have been accumulated by genome anal. An extremely effective means is provided by this type of protein-labeling compound from the stand point of an efficiency or automation in the functional anal. of proteins such as nucleic acid-protein interaction or protein-protein interaction. As examples, ribocytidyl(3'→5')puromycin, deoxyuridyl(3'→5')puromycin, deoxycytidyl(3'→5')puromycin, Fluorpur and Fluorhiopur were synthesized and effectively used for the labeling of **thioredoxin expressed** in E. coli cell-free extract

L9 ANSWER 28 OF 37 BIOTECHDS COPYRIGHT 2006 THE THOMSON CORP. on STN  
DUPLICATE 1

ACCESSION NUMBER: 1998-09126 BIOTECHDS

TITLE: Production of polypeptides as inclusion bodies;  
recombinant protein preparation by plasmid  
pTrc99A or plasmid pTrxfus vector-mediated  
**thioredoxin expression** in Escherichia  
coli inclusion body

AUTHOR: Chatterjee D; Longo M; Flynn E; Oberfelder R

PATENT ASSIGNEE: Life-Technol.

LOCATION: Rockville, MD, USA.

PATENT INFO: WO 9830684 16 Jul 1998

APPLICATION INFO: WO 1998-US492 8 Jan 1998

PRIORITY INFO: US 1997-34658 8 Jan 1997

DOCUMENT TYPE: Patent

LANGUAGE: English

OTHER SOURCE: WPI: 1998-399134 [34]

AB A new method for the preparation of a protein in the form of an inclusion body involves: obtaining a host cell (e.g. Escherichia coli) containing a first DNA sequence encoding the protein linked to a second DNA sequence encoding an inclusion partner (**thioredoxin** or modified **thioredoxin**), forming a gene fusion construct (plasmid pTrcpr1-monomer or pTrxA-concat); and culturing the cell to favor production of the protein as inclusion bodies. Also new are: vector plasmid pTrc99A and plasmid pTrxfus containing the construct; a host cell containing the vector; making a protein mol.weight ladder composition by obtaining one or more DNA sequences encoding proteins of different mol.weight values, transforming host cells with the DNA, culturing the cells to favor production of each protein, and isolating each protein; and making one or more stained proteins by incubating the proteins with one or more protein-binding **dyes** under incubation conditions to complex the proteins with the **dyes**. The methods may be used to prepare a fragment of the gene-32 protein of phage T4, a fragment of KpnI-methylase or a fragment of E. coli Dead-Box protein or **thioredoxin**. (84pp)

L9 ANSWER 29 OF 37 MEDLINE on STN DUPLICATE 2

ACCESSION NUMBER: 1998288331 MEDLINE

DOCUMENT NUMBER: PubMed ID: 9624185

TITLE: Disruption of redox homeostasis in the transforming growth factor-alpha/c-myc transgenic mouse model of accelerated hepatocarcinogenesis.

AUTHOR: Factor V M; Kiss A; Woitach J T; Wirth P J; Thorgeirsson S  
 CORPORATE SOURCE: Laboratory of Experimental Carcinogenesis, NCI, National  
 Institutes of Health, Bethesda, Maryland 20892, USA.  
 SOURCE: The Journal of biological chemistry, (1998 Jun 19) Vol.  
 273, No. 25, pp. 15846-53.  
 Journal code: 2985121R. ISSN: 0021-9258.  
 PUB. COUNTRY: United States  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 199807  
 ENTRY DATE: Entered STN: 19980716  
 Last Updated on STN: 19980716  
 Entered Medline: 19980709

AB In previous studies we have demonstrated that transforming growth factor  
 (TGF)-alpha/c-myc double transgenic mice exhibit an enhanced rate of cell  
 proliferation, accumulate extensive DNA damage, and develop multiple liver  
 tumors between 4 and 8 months of age. To clarify the biochemical events  
 that may be responsible for the genotoxic and carcinogenic effects  
 observed in this transgenic model, several parameters of redox homeostasis  
 in the liver were examined prior to development of hepatic tumors. By 2  
 months of age, production of reactive oxygen species, determined by the  
 peroxidation-sensitive fluorescent dye, 2',7'-dichlorofluorescein  
 diacetate, was significantly elevated in TGF-alpha/c-myc transgenic  
 hepatocytes versus either wild type or c-myc single transgenic cells, and  
 occurred in parallel with an increase in lipid peroxidation.  
 Concomitantly with a rise in oxidant levels, antioxidant defenses were  
 decreased, including total glutathione content and the activity of  
 glutathione peroxidase, whereas thioredoxin reductase activity  
 was not changed. However, hepatic tumors which developed in  
 TGF-alpha/c-myc mice exhibited an increase in thioredoxin  
 reductase activity and a very low activity of glutathione peroxidase.  
 Furthermore, specific deletions were detected in mtDNA as early as 5 weeks  
 of age in the transgenic mice. These data provide experimental evidence  
 that co-expression of TGF-alpha and c-myc transgenes in mouse  
 liver promotes overproduction of reactive oxygen species and thus creates  
 an oxidative stress environment. This phenomenon may account for the  
 massive DNA damage and acceleration of hepatocarcinogenesis observed in  
 the TGF-alpha/c-myc mouse model.

L9 ANSWER 30 OF 37 MEDLINE on STN  
 ACCESSION NUMBER: 1998181877 MEDLINE  
 DOCUMENT NUMBER: PubMed ID: 9523703  
 TITLE: Characterization of wheat thioredoxin h cDNA and  
 production of an active Triticum aestivum protein in  
 Escherichia coli.  
 AUTHOR: Gautier M F; Lullien-Pellerin V; de Lamotte-Guery F; Guirao  
 A; Joudrier P  
 CORPORATE SOURCE: Unite de Biochimie et Biologie Moleculaire des Cereales,  
 INRA, Montpellier, France.. gautier@ensam.inra.fr  
 SOURCE: European journal of biochemistry / FEBS, (1998 Mar 1) Vol.  
 252, No. 2, pp. 314-24.  
 Journal code: 0107600. ISSN: 0014-2956.  
 PUB. COUNTRY: GERMANY: Germany, Federal Republic of  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 OTHER SOURCE: GENBANK-AJ001903; GENBANK-X69915  
 ENTRY MONTH: 199804  
 ENTRY DATE: Entered STN: 19980430  
 Last Updated on STN: 20000303  
 Entered Medline: 19980423

AB Two cDNA clones, pTam13.38 and pTd14.13.2, encoding a Triticum

aestivum and a *Triticum durum* thioredoxin h, respectively, were isolated from mid-maturation seed cDNA libraries. The T aestivum thioredoxin h has a molecular mass of 13.5 kDa and that from T durum has a molecular mass of 13.8 kDa. These two wheat thioredoxin h are 98.5% similar and contain the canonical WCGPC active site and the important structural and functional amino acids that are conserved in thioredoxin sequences. The recombinant T. aestivum thioredoxin h (TrxTa) overproduced in BL21(DE3)pLysS was purified to homogeneity by a three-step procedure including heat treatment, anion-exchange chromatography and gel filtration. TrxTa showed a lower stability to high temperature than *Escherichia coli* thioredoxin or plant thioredoxin m. The molecular mass of TrxTa, determined by mass spectrometry, is 13,391 Da and corresponds to a protein lacking the first methionine residue, as confirmed by its N-terminal end sequence AASAAT. Using the 5,5'-dithiobis(2-nitrobenzoic acid)-reduction assay and monobromobimane revelation we showed that TrxTa is specifically reduced by wheat NADP:thioredoxin reductase (NTR), and not by E. coli NTR. TrxTa is able to reduce identified target proteins i.e. wheat seed alpha-amylase inhibitors (chloroform/methanol-soluble proteins). The presence of a putative transmembrane domain at the N-terminal end of the two wheat thioredoxins raises the question of whether these proteins are membrane anchored.

L9 ANSWER 31 OF 37 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 1999:63389 BIOSIS

DOCUMENT NUMBER: PREV199900063389

TITLE: Increased expression of annexin I and thioredoxin detected by two-dimensional gel electrophoresis of drug resistant human stomach cancer cells.

AUTHOR(S): Sinha, Pranav [Reprint author]; Huetter, Gero; Koettgen, Eckart; Dietel, Manfred; Schadendorf, Dirk; Lage, Hermann  
CORPORATE SOURCE: Inst. Laboratoriumsmed. und Pathobiochem., Campus Virchow-Klinikum, Universitaetsklin. Charite, Augustenburger Platz 1, Berlin, Germany

SOURCE: Journal of Biochemical and Biophysical Methods, (Nov. 18, 1998) Vol. 37, No. 3, pp. 105-116. print.  
CODEN: JBBMDG. ISSN: 0165-022X.

DOCUMENT TYPE: Article

LANGUAGE: English

ENTRY DATE: Entered STN: 16 Feb 1999

Last Updated on STN: 16 Feb 1999

AB The therapy of advanced cancer using chemotherapy alone or in combination with radiation or hyperthermia yields an overall response rate of about 20-50%. This success is often marred by the development of resistance to cytostatic drugs. Our aim was to study the global analysis of protein expression in the development of chemoresistance in vitro. We therefore used a cell culture model derived from the gastric carcinoma cell line EPG 85-257P. A classical multidrug-resistant subline EPG85-257RDB selected to daunorubicin and an atypical multidrug-resistant cell variant EPG85-257RNOV selected to mitoxantrone, were analysed using two-dimensional electrophoresis in immobilized pH-gradients (pH 4.0-8.0) in the first dimension and linear polyacrylamide gels (12%) in the second dimension. After staining with coomassie brilliant blue, image analysis was performed using the PDQuest system. Spots of interest were isolated using preparative two-dimensional electrophoresis and subjected to microsequencing. A total of 241 spots from the EPG85-257RDB-standard and 289 spots from the EPG85-257RNOV-standard could be matched to the EPG85-257P-standard. Microsequencing after enzymatic hydrolysis in gel, mass spectrometric data and sequencing of the peptides after their fractionation using microbore HPLC identified that two proteins annexin I and thioredoxin were overexpressed in chemoresistant cell lines. Annexin I was present in both the classical and the atypical

multidrug-resistant cells. Thioredoxin was found to be overexpressed only in the atypical multidrug-resistant cell line.

L9 ANSWER 32 OF 37 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1997:585491 HCAPLUS

DOCUMENT NUMBER: 128:44390

TITLE: Complete genome sequence of Escherichia coli K-12

AUTHOR(S): Blattner, Frederick R.; Plunkett, Guy, III; Bloch, Craig A.; Perna, Nicole T.; Burland, Valerie; Riley, Monica; Collado-Vides, Julio; Glasner, Jeremy D.; Rode, Christopher K.; Mayhew, George F.; Gregor, Jason; Davis, Nelson Wayne; Kirkpatrick, Heather A.; Goeden, Michael A.; Rose, Debra J.; Mau, Bob; Shao, Ying

CORPORATE SOURCE: Lab. Genetics, Univ. Wisconsin-Madison, Madison, WI, 53706, USA

SOURCE: Science (Washington, D. C.) (1997), 277(5331), 1453-1462

CODEN: SCIEAS; ISSN: 0036-8075

PUBLISHER: American Association for the Advancement of Science

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The 4,639,221-base pair sequence of Escherichia coli K-12 is presented. Of 4288 protein-coding genes annotated, 38 percent have no attributed function. Comparison with five other sequenced microbes reveals ubiquitous as well as narrowly distributed gene families; many families of similar genes within E. coli are also evident. The largest family of paralogous proteins contains 80 ABC transporters. The genome as a whole is strikingly organized with respect to the local direction of replication; guanines, oligonucleotides possibly related to replication and recombination, and most genes are so oriented. The genome also contains insertion sequence (IS) elements, phage remnants, and many other patches of unusual composition indicating genome plasticity through horizontal transfer.

REFERENCE COUNT: 98 THERE ARE 98 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 33 OF 37 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1997:661238 HCAPLUS

DOCUMENT NUMBER: 127:304503

TITLE: Structures and functions of the two proteins in bovine adrenocortical mitochondria. A unique defense system against reactive oxygen species

AUTHOR(S): Hiroi, Tomoko

CORPORATE SOURCE: Radioisotope Research Institute Basic Medicine, St. Marianna University School Medicine, Kawasaki, 216, Japan

SOURCE: Sei Marianna Ika Daigaku Zasshi (1997), 25(3), 183-198  
CODEN: SMIZDS; ISSN: 0387-2289

PUBLISHER: Sei-Marianna Ika Daigaku Igakkai

DOCUMENT TYPE: Journal

LANGUAGE: Japanese

AB The present author's group has reported previously that a unique protein (Mr 22,000 dalton) originally detected in the bovine adrenal cortex is a substrate protein (SP-22) for bovine ATP-dependent protease. Both SP-22 and the protease are localized in the mitochondrial matrix of bovine adrenal cortex. A third protein termed SP-22 activating protein (SAP) that activates a novel, reducing activity of SP-22 has also been found in the bovine adrenocortical mitochondria. To study the possible, biochem. interrelationship among these proteins, the present author utilized techniques of mol. biol. Firstly, oligonucleotide primers were prepared for SP-22 and SAP based on amino acid sequences for their oligopeptides. Methods used for isolation of cDNAs were reverse transcription-polymerase chain reaction, screening of bovine adrenal medulla cDNA library, and

cloning by the rapid amplification of cDNA ends methods. Secondly, nucleotide sequences of the selected clones were determined by the dye-labeled dideoxy chain termination method in an automatic DNA sequencer. The entire nucleotide sequences determined for SP-22 and SAP were 1646 bp and 1040 bp long, resp. Amino acid sequences deduced from these nucleotide sequences revealed that both SP-22 and SAP contain mitochondrial targeting signals of 62 residues and 59 residues, resp. It was further identified that these signals are processed during their transportation into mitochondria. Both nucleotide- and amino acid sequences were compared with those of various proteins in the seven different com. databases. Protein SP-22 showed a high homol. with antioxidant protein family enzymes while SAP was completely homologous to the mammalian mitochondrial thioredoxin. Considering the chemical properties, we concluded that SP-22 is a thioredoxin-dependent peroxide reductase. Thus, the present lines of evidence indicate the presence of a new antioxidant system functioning in the mitochondria of the adrenal cortex as well as of the other tissues. In summary, this particular thioredoxin dependent system reduces hydrogen peroxide, a hazardous byproduct of the steroid-hydroxylases in the adrenal cortex as well as of Mn-superoxide dismutase in other tissues. Intramitochondrial levels of reactive oxygen species are regulated by a combined function of the ATP-dependent protease, SP-22 and SAP.

L9 ANSWER 34 OF 37 MEDLINE on STN  
 ACCESSION NUMBER: 97415331 MEDLINE  
 DOCUMENT NUMBER: PubMed ID: 9271130  
 TITLE: Identification of a recombinant synaptobrevin-thioredoxin fusion protein by capillary zone electrophoresis using laser-induced fluorescence detection.  
 AUTHOR: Asermely K E; Broomfield C A; Nowakowski J; Courtney B C; Adler M  
 CORPORATE SOURCE: Pharmacology Division, U.S. Army Medical Research Institute of Chemical Defense, Aberdeen Proving Ground, MD 21010-5425, USA.  
 SOURCE: Journal of chromatography. B, Biomedical sciences and applications, (1997 Jul 18) Vol. 695, No. 1, pp. 67-75. Journal code: 9714109. ISSN: 1387-2273.  
 PUB. COUNTRY: Netherlands  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 199709  
 ENTRY DATE: Entered STN: 19971008  
 Last Updated on STN: 19980206  
 Entered Medline: 19970925

AB Capillary zone electrophoresis (CZE) was utilized to identify a synaptobrevin-thioredoxin fusion protein (TSB-51). TSB-51 is a substrate for cleavage by botulinum toxin B at the Q(76)-F(77) site. TSB-51 was derivatized with a fluorophore, CBQCA [3-(4-carboxy-benzoyl)-2-quinoline-carboxaldehyde], for 4 h at room temperature. Optimal conditions for CZE separation of the TSB-51-CBQCA complex were determined: buffer (sodium borate), pH (9.0), applied voltage (25 kV), temperature (25 degrees C) and forward polarity. SDS-PAGE showed that TSB-51 had a molecular mass of approximately 19 kDa. The protein was transferred to PVDF membrane and sequenced by the Edman degradation method verifying the first twelve amino acids as SDKIIHLTDDSF. TSB-51 was also collected during CZE separation and subsequently sequenced yielding the first three amino acids as SDK. This CZE-LIF method coupled with the CBQCA derivatization, fraction collection and Edman sequencing allowed for identification of the recombinant protein, a fast separation run time and utilization of small volumes of peptide (1.5 ng protein/23.6 nl injection). This method will be used for monitoring the endopeptidase activity of botulinum toxin B on TSB-51.

L9 ANSWER 35 OF 37 MEDLINE on STN DUPLICATE 3  
 ACCESSION NUMBER: 96198118 MEDLINE  
 DOCUMENT NUMBER: PubMed ID: 8626451  
 TITLE: Efficient expression of the gene for spinach phosphoribulokinase in *Pichia pastoris* and utilization of the recombinant enzyme to explore the role of regulatory cysteinyl residues by site-directed mutagenesis.  
 AUTHOR: Brandes H K; Hartman F C; Lu T Y; Larimer F W  
 CORPORATE SOURCE: Protein Engineering Program, Biology Division, Oak Ridge National Laboratory, Tennessee 37831, USA.  
 SOURCE: The Journal of biological chemistry, (1996 Mar 15) Vol. 271, No. 11, pp. 6490-6.  
 Journal code: 2985121R. ISSN: 0021-9258.  
 PUB. COUNTRY: United States  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 199606  
 ENTRY DATE: Entered STN: 19960708  
 Last Updated on STN: 20000303  
 Entered Medline: 19960624

AB Phosphoribulokinase (PRK), unique to photosynthetic organisms, is regulated in higher plants by thioredoxin-mediated thiol-disulfide exchange in a light-dependent manner. Prior attempts to overexpress the higher plant PRK gene in *Escherichia coli* for structure-function studies have been hampered by sensitivity of the recombinant protein to proteolysis as well as toxic effects of the protein on the host. To overcome these impediments, we have spliced the spinach PRK coding sequence immediately downstream from the AOX1 (alcohol oxidase) promoter of *Pichia pastoris*, displacing the chromosomal AOX1 gene. The PRK gene is now expressed, in response to methanol, at 4-6% of total soluble protein, without significant in vivo degradation of the recombinant enzyme. This recombinant spinach PRK is purified to homogeneity by successive anion-exchange and dye-affinity chromatography and is shown to be electrophoretically and kinetically indistinguishable from the authentic spinach counterpart. Site-specific replacement of all of PRK's cysteinyl residues (both individually and in combination) demonstrates a modest catalytically facilitative role for Cys-55 (one of the regulatory residues) and the lack of any catalytic role for Cys-16 (the other regulatory residue), Cys-244, or Cys-250. Mutants with seryl substitutions at position 55 display non-hyperbolic kinetics relative to the concentration of ribulose 5-phosphate. Sulfate restores hyperbolic kinetics and enhances kinase activity, presumably reflecting conformational differences between the position 55 mutants and wild-type enzyme. Catalytic competence of the C16S-C55S double mutant proves that mere loss of free sulphydryl groups by oxidative regulation cannot account entirely for the accompanying total inactivation.

L9 ANSWER 36 OF 37 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN  
 ACCESSION NUMBER: 1995:747446 SCISEARCH  
 THE GENUINE ARTICLE: TB584  
 TITLE: PURIFICATION, CHARACTERIZATION, AND CLONING OF A HEME-BINDING PROTEIN (23 KDA) IN RAT-LIVER CYTOSOL  
 AUTHOR: IWAHARA S (Reprint); SATOH H; SONG D X; WEBB J; BURLINGAME A L; NAGAE Y; MULLEREBERHARD U  
 CORPORATE SOURCE: CORNELL UNIV, COLL MED, DEPT PEDIAT, NEW YORK, NY 10021;  
 CORNELL UNIV, COLL MED, DEPT BIOCHEM, NEW YORK, NY 10021;  
 CORNELL UNIV, COLL MED, DEPT PHARMACOL, NEW YORK, NY 10021;  
 ILLINOIS STATE UNIV, DEPT CHEM, NORMAL, IL 61790;  
 UNIV CALIF SAN FRANCISCO, DEPT PHARMACEUT CHEM, SAN FRANCISCO, CA 94143;  
 UNIV CALIF SAN FRANCISCO, CTR LIVER, SAN FRANCISCO, CA 94143;  
 NIPPON MED COLL, DEPT INTERNAL

MED, TOKYO 113, JAPAN  
COUNTRY OF AUTHOR: USA; JAPAN  
SOURCE: BIOCHEMISTRY, (17 OCT 1995) Vol. 34, No. 41, pp.  
13398-13406.  
ISSN: 0006-2960.  
PUBLISHER: AMER CHEMICAL SOC, PO BOX 57136, WASHINGTON, DC 20037-0136  
DOCUMENT TYPE: Article; Journal  
FILE SEGMENT: LIFE  
LANGUAGE: English  
REFERENCE COUNT: 58  
ENTRY DATE: Entered STN: 1995  
Last Updated on STN: 1995

\*ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS\*

AB A heme-binding protein (designated HBP23) has been purified from rat liver cytosol using heme-affinity chromatography and either reverse-phase high-performance liquid chromatography or sequential ion-exchange chromatography. The protein (23 kDa) binds heme with an affinity ( $K_d = 55$  nM) higher than that of the abundant cytosolic heme-binding proteins, heme-binding protein (HBP)/liver fatty acid-binding protein (L-FABP) and the glutathione S-transferases (GSTs) ( $K_d = 100$ -200 nM). HBP23 is present in the cytosol of liver, kidney, spleen, small intestine, and heart, with the liver showing the highest content. A cDNA coding the 23-kDa protein was cloned using reverse transcription polymerase chain reaction with degenerative oligonucleotides derived from partial amino acid sequences. The cloned cDNA encoded 199 amino acids, and its amino acid sequence showed no homology to HBP/L-FABP, GSTs, or any other heme-binding proteins or hemoproteins. Homology search showed that HBP23 is highly homologous to mouse macrophage 23-kDa stress protein, which is inducible by oxidant stress in peritoneal macrophages [Ishii, T., Yamada, M., Sate, H., Matsue, M., Taketani, S., Nakayama, K., Sugita, Y., and Bannai, S. (1993) J. Biol. Chemical 268, 18633-18636]. Thioredoxin peroxidase as well as HBP23 and the mouse macrophage 23-kDa stress protein are members of the peroxiredoxin family, a recently recognized class of antioxidant proteins [Chae, H. Z., Chung, S. J., & Rhee, S. G. (1994) J. Biol. Chemical 269, 27670-27678]. An increase in HBP23 mRNA was observed in Hepa 1-6 cells after treatment with heme and cadmium and during liver regeneration after partial hepatectomy. These findings indicate an involvement of HBP23 in heme metabolism.

L9 ANSWER 37 OF 37 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 1991:316120 SCISEARCH

THE GENUINE ARTICLE: FP306

TITLE: ESCHERICHIA-COLI GLUTAREDOXIN - CLONING AND  
OVEREXPRESSION, THERMODYNAMIC STABILITY OF THE OXIDIZED  
AND REDUCED FORMS, AND REPORT OF AN N-TERMINAL EXTENDED  
SPECIES

AUTHOR: SANDBERG V A (Reprint); KREN B; FUCHS J A; WOODWARD C

CORPORATE SOURCE: UNIV MINNESOTA, DEPT BIOCHEM, ST PAUL, MN 55108

COUNTRY OF AUTHOR: USA

SOURCE: BIOCHEMISTRY, (4 JUN 1991) Vol. 30, No. 22, pp. 5475-5484.  
ISSN: 0006-2960.

PUBLISHER: AMER CHEMICAL SOC, 1155 16TH ST, NW, WASHINGTON, DC 20036.

DOCUMENT TYPE: Article; Journal

FILE SEGMENT: LIFE

LANGUAGE: English

REFERENCE COUNT: 67

ENTRY DATE: Entered STN: 1994

Last Updated on STN: 1994

\*ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS\*

AB Escherichia coli glutaredoxin (MW 9700) catalyzes intracellular redox reactions utilizing a disulfide/dithiol enzymatic mechanism involving the active-site residues -Cys-Pro-Tyr-Cys-. It is functionally related to the



thioredoxin family and is expected to share similar three-dimensional structure [Eklund, H., Cambillau, C., Sjoberg, B.-M., Holmgren, A., Jornvall, H., Hoog, J.-O., & Branden, C.-I. (1984) EMBO J. 3, 1443-1449]. We constructed an overexpression system in which production of glutaredoxin is controlled by temperature-sensitive expression of the phage T7 promoter. In addition to glutaredoxin extended by the sequence Met-Arg-Arg-Glu-Ile- at the N terminus. We have begun characterization of the structure and stability of the oxidized and reduced forms of glutaredoxin (grx-S2 and grx-(SH)<sub>2</sub>, respectively). Secondary structure calculated from CD data agrees with that predicted from the three-dimensional model of Eklund et al. The cooperative denaturation reactions of oxidized and reduced glutaredoxin were measured in temperature-induced and guanidine hydrochloride induced unfolding experiments. Surprisingly, oxidized and reduced glutaredoxins are very similar in stability. In heat-induced denaturation, monitored by CD, T<sub>m</sub> is 55 and 57-degrees-C for oxidized and reduced, respectively. In GuHCl denaturation, monitored by fluorescence, the midpoint denaturant concentrations are 2 M for both oxidized and reduced. It follows that the redox potentials of the disulfide bond are similar in unfolded and folded glutaredoxin. This is unexpected because in *E. coli* thioredoxin the oxidized form is far more stable than the reduced [Kelley, R. F., Shalongo, W., Jagannadham, M. V., & Stellwagen, E. (1987) Biochemistry 26, 1406-1411] and the redox potential of folded thioredoxin is significantly more negative than that of unfolded thioredoxin [Lin, T.-Y., & Kim, P. (1989) Biochemistry 28, 5282-5287].

=> d his

(FILE 'HOME' ENTERED AT 14:18:13 ON 11 APR 2006)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS, LIFESCI' ENTERED AT 14:18:37 ON 11 APR 2006

```
L1      25776 S THIOREDOXIN
L2      7634805 S CLON? OR EXPRESS? OR RECOMBINANT
L3      12217 S L1 AND L2
L4      7209 S HISTIDINE (W) TAG?
L5      83 S L3 AND L4
L6      662353 S DYE?
L7      0 S L5 AND L6
L8      48 S L3 AND L6
L9      37 DUP REM L8 (11 DUPLICATES REMOVED)
```

=> s inclusion (w) bod?

```
L10     50437 INCLUSION (W) BOD?
```

=> s l9 and l10

```
L11     1 L9 AND L10
```

=> d all

```
L11     ANSWER 1 OF 1 BIOTECHDS COPYRIGHT 2006 THE THOMSON CORP. on STN
AN      1998-09126 BIOTECHDS
TI      Production of polypeptides as inclusion bodies;
         recombinant protein preparation by plasmid pTrc99A or
         plasmid pTrxfus vector-mediated thioredoxin
         expression in Escherichia coli inclusion
         body
AU      Chatterjee D; Longo M; Flynn E; Oberfelder R
PA      Life-Technol.
LO      Rockville, MD, USA.
PI      WO 9830684 16 Jul 1998
AI      WO 1998-US492 8 Jan 1998
PRAI    US 1997-34658 8 Jan 1997
```

DT Patent  
 LA English  
 OS WPI: 1998-399134 [34]  
 AB A new method for the preparation of a protein in the form of an inclusion body involves: obtaining a host cell (e.g. Escherichia coli) containing a first DNA sequence encoding the protein linked to a second DNA sequence encoding an inclusion partner (thioredoxin or modified thioredoxin), forming a gene fusion construct (plasmid pTrcpr1-monomer or pTrxA-concat); and culturing the cell to favor production of the protein as inclusion bodies. Also new are: vector plasmid pTrc99A and plasmid pTrxfus containing the construct; a host cell containing the vector; making a protein mol.weight ladder composition by obtaining one or more DNA sequences encoding proteins of different mol.weight values, transforming host cells with the DNA, culturing the cells to favor production of each protein, and isolating each protein; and making one or more stained proteins by incubating the proteins with one or more protein-binding dyes under incubation conditions to complex the proteins with the dyes. The methods may be used to prepare a fragment of the gene-32 protein of phage T4, a fragment of KpnI-methylase or a fragment of E. coli Dead-Box protein or thioredoxin. (84pp)

CC D PHARMACEUTICALS; D3 Peptides and Proteins; A GENETIC ENGINEERING AND FERMENTATION; A1 Nucleic Acid Technology; L PURIFICATION; L1 Downstream Processing

CT PHAGE T4 GENE-32 PROTEIN, KPN1 DNA-METHYLASE, ESCHERICHIA COLI DEAD-BOX PROTEIN, THIOREDOXIN RECOMBINANT FUSION PROTEIN PREP., PROTEIN PURIFICATION, VECTOR PLASMID PTRCPRI-MONOMER, PLASMID PTRXA-CONCAT EXPRESSION IN ESCHERICHIA COLI INCLUSION BODY, APPL. MOL.WT. LADDER CONSTRUCTION, PROTEIN DYEING ENZYME DNA SEQUENCE PROTEIN SEQUENCE BACTERIUM CLONING (VOL.17, NO.21)

=> d his

(FILE 'HOME' ENTERED AT 14:18:13 ON 11 APR 2006)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS, LIFESCI' ENTERED AT 14:18:37 ON 11 APR 2006

L1 25776 S THIOREDOXIN  
 L2 7634805 S CLON? OR EXPRESS? OR RECOMBINANT  
 L3 12217 S L1 AND L2  
 L4 7209 S HISTIDINE (W) TAG?  
 L5 83 S L3 AND L4  
 L6 662353 S DYE?  
 L7 0 S L5 AND L6  
 L8 48 S L3 AND L6  
 L9 37 DUP REM L8 (11 DUPLICATES REMOVED)  
 L10 50437 S INCLUSION (W) BOD?  
 L11 1 S L9 AND L10

=> e chatterjee d k/au

E1 1 CHATTERJEE D E B/AU  
 E2 36 CHATTERJEE D J/AU  
 E3 381 --> CHATTERJEE D K/AU  
 E4 54 CHATTERJEE D N/AU  
 E5 23 CHATTERJEE D P/AU  
 E6 3 CHATTERJEE D R/AU  
 E7 55 CHATTERJEE D S/AU  
 E8 7 CHATTERJEE DANIEL/AU  
 E9 5 CHATTERJEE DAS J M/AU  
 E10 16 CHATTERJEE DAS S/AU  
 E11 1 CHATTERJEE DAS SASWATI/AU  
 E12 5 CHATTERJEE DEB/AU

```
=> s e3-e12
L12      550 ("CHATTERJEE D K"/AU OR "CHATTERJEE D N"/AU OR "CHATTERJEE D
          P"/AU OR "CHATTERJEE D R"/AU OR "CHATTERJEE D S"/AU OR "CHATTERJ
          EE DANIEL"/AU OR "CHATTERJEE DAS J M"/AU OR "CHATTERJEE DAS S"/AU
          OR "CHATTERJEE DAS SASWATI"/AU OR "CHATTERJEE DEB"/AU)
```

```
=> e longo m/au
E1        15      LONGO LUIGIA/AU
E2         6      LONGO LUIZ S JR/AU
E3       698 --> LONGO M/AU
E4       113      LONGO M A/AU
E5         9      LONGO M B/AU
E6        23      LONGO M C/AU
E7         2      LONGO M CASTELLANI/AU
E8        14      LONGO M D/AU
E9         2      LONGO M D C/AU
E10       20      LONGO M F/AU
E11       20      LONGO M G/AU
E12       15      LONGO M I/AU
```

```
=> s e3
L13      698 "LONGO M"/AU
```

```
=> e oberfelder r/au
E1         1      OBERFELD SHELDON M/AU
E2         1      OBERFELDARZT/AU
E3        14 --> OBERFELDER R/AU
E4        30      OBERFELDER R W/AU
E5         6      OBERFELDER ROBERT/AU
E6         5      OBERFELDER ROBERT W/AU
E7         1      OBERFELDER ROBERT WILLIAM/AU
E8        15      OBERFELL C R/AU
E9        10      OBERFELL CHARLES R/AU
E10        1      OBERFELL CHAS R/AU
E11       73      OBERFELL G G/AU
E12        1      OBERFELL G S/AU
```

```
=> s e3-e7
L14      56 ("OBERFELDER R"/AU OR "OBERFELDER R W"/AU OR "OBERFELDER ROBERT"
          /AU OR "OBERFELDER ROBERT W"/AU OR "OBERFELDER ROBERT WILLIAM"/A
          U)
```

```
=> e flynn e/au
E1         1      FLYNN DUANE J/AU
E2         3      FLYNN DUSTIE/AU
E3       285 --> FLYNN E/AU
E4        54      FLYNN E A/AU
E5         1      FLYNN E B/AU
E6         5      FLYNN E C/AU
E7         9      FLYNN E D/AU
E8         3      FLYNN E E/AU
E9         1      FLYNN E F/AU
E10        11      FLYNN E H/AU
E11       151      FLYNN E J/AU
E12        1      FLYNN E J */AU
```

```
=> s e3
L15      285 "FLYNN E"/AU
```

```
=> d his
```

(FILE 'HOME' ENTERED AT 14:18:13 ON 11 APR 2006)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS,  
LIFESCI' ENTERED AT 14:18:37 ON 11 APR 2006

L1 25776 S THIOREDOXIN  
L2 7634805 S CLON? OR EXPRESS? OR RECOMBINANT  
L3 12217 S L1 AND L2  
L4 7209 S HISTIDINE (W) TAG?  
L5 83 S L3 AND L4  
L6 662353 S DYE?  
L7 0 S L5 AND L6  
L8 48 S L3 AND L6  
L9 37 DUP REM L8 (11 DUPLICATES REMOVED)  
L10 50437 S INCLUSION (W) BOD?  
L11 1 S L9 AND L10  
E CHATTERJEE D K/AU  
L12 550 S E3-E12  
E LONGO M/AU  
L13 698 S E3  
E OBERFELDER R/AU  
L14 56 S E3-E7  
E FLYNN E/AU  
L15 285 S E3

=> s l11 or l12 or l13 or l14 or l15

L16 1578 L11 OR L12 OR L13 OR L14 OR L15

=> s l9 and l16

L17 1 L9 AND L16

=> d ibib ab

L17 ANSWER 1 OF 1 BIOTECHDS COPYRIGHT 2006 THE THOMSON CORP. on STN

ACCESSION NUMBER: 1998-09126 BIOTECHDS

TITLE: Production of polypeptides as inclusion  
bodies;

recombinant protein preparation by plasmid  
pTrc99A or plasmid pTrxfus vector-mediated  
thioredoxin expression in Escherichia  
coli inclusion body

AUTHOR: Chatterjee D; Longo M; Flynn E;  
Oberfelder R

PATENT ASSIGNEE: Life-Technol.

LOCATION: Rockville, MD, USA.

PATENT INFO: WO 9830684 16 Jul 1998

APPLICATION INFO: WO 1998-US492 8 Jan 1998

PRIORITY INFO: US 1997-34658 8 Jan 1997

DOCUMENT TYPE: Patent

LANGUAGE: English

OTHER SOURCE: WPI: 1998-399134 [34]

AB A new method for the preparation of a protein in the form of an  
inclusion body involves: obtaining a host cell (e.g.  
Escherichia coli) containing a first DNA sequence encoding the protein  
linked to a second DNA sequence encoding an inclusion partner (  
thioredoxin or modified thioredoxin), forming a gene  
fusion construct (plasmid pTrcpr1-monomer or pTrxA-concat); and culturing  
the cell to favor production of the protein as inclusion  
bodies. Also new are: vector plasmid pTrc99A and plasmid pTrxfus  
containing the construct; a host cell containing the vector; making a  
protein mol.weight ladder composition by obtaining one or more DNA sequences  
encoding proteins of different mol.weight values, transforming host cells  
with the DNA, culturing the cells to favor production of each protein,  
and isolating each protein; and making one or more stained proteins by  
incubating the proteins with one or more protein-binding dyes  
under incubation conditions to complex the proteins with the dyes  
. The methods may be used to prepare a fragment of the gene-32 protein

of phage T4, a fragment of KpnI-methylase or a fragment of E. coli  
Dead-Box protein or thioredoxin. (84pp)

=> d his

(FILE 'HOME' ENTERED AT 14:18:13 ON 11 APR 2006)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS,  
LIFESCI' ENTERED AT 14:18:37 ON 11 APR 2006

L1	25776 S THIOREDOXIN
L2	7634805 S CLON? OR EXPRESS? OR RECOMBINANT
L3	12217 S L1 AND L2
L4	7209 S HISTIDINE (W) TAG?
L5	83 S L3 AND L4
L6	662353 S DYE?
L7	0 S L5 AND L6
L8	48 S L3 AND L6
L9	37 DUP REM L8 (11 DUPLICATES REMOVED)
L10	50437 S INCLUSION (W) BOD?
L11	1 S L9 AND L10
	E CHATTERJEE D K/AU
L12	550 S E3-E12
	E LONGO M/AU
L13	698 S E3
	E OBERFELDER R/AU
L14	56 S E3-E7
	E FLYNN E/AU
L15	285 S E3
L16	1578 S L11 OR L12 OR L13 OR L14 OR L15
L17	1 S L9 AND L16

	Issue Date	Pages	Document ID	Title
1	20060406	70	US 2006007353 0 A1	Methods and compositions involving sortase B
2	20060216	83	US 2006003531 5 A1	Transporters and ion channels
3	20051020	22	US 2005023335 7 A1	Linking gene sequence to gene function by three dimensional (3D) protein structure determination
4	20050526	32	US 2005011263 7 A1	Mutant DNA polymerases and uses thereof
5	20050127	39	US 2005001982 9 A1	Protein/solubility folding assessed by structural complementation
6	20041216	117	US 2004025370 1 A1	Protein and peptide fragments from mouse telomerase reverse transcriptase
7	20041209	144	US 2004024825 1 A1	Receptors and membrane associated proteins
8	20041111	171	US 2004022491 1 A1	Transporters and ion channels
9	20041014	31	US 2004020456 3 A1	Methods for production of proteins
10	N/A, Contact help desk	31	US 2004015729 1 A1	
11	N/A, Contact help desk	72	US 2004015287 4 A1	
12	N/A, Contact help desk	46	US 2004012768 3 A1	

13	20040617	140	US 2004011666 6 A1	Transporters and ion channels
----	----------	-----	--------------------------	----------------------------------

	Issue Date	Pages	Document ID	Title
14	20040318	182	US 20040053258 A1	Transporters and ion channels
15	20040226	87	US 20040037846 A1	Chlamydia pmp proteins, gene sequences and uses thereof
16	20040205	140	US 20040024183 A1	Transporters and ion channels
17	20040205	73	US 20040023207 A1	Assays for drug discovery based on microcompetition with a foreign polynucleotide
18	20040205	72	US 20040023206 A1	Methods for chronic disease diagnosis based on microcompetition with a foreign polynucleotide
19	20040205	63	US 20040022764 A1	Inhibition of microcompetition with a foreign polynucleotide as treatment of chronic disease
20	20040122	119	US 20040014945 A1	Transporters and ion channels
21	20040101	40	US 20040002065 A1	PROTEIN/SOLUBILITY FOLDING ASSESSED BY STRUCTURAL COMPLEMENTATION
22	20031120	144	US 20030216310 A1	Transporters and ion channels
23	20031120	43	US 20030215915 A1	Cytochrome P450 expression in enterobacteria
24	20031113	94	US 20030211499 A1	Transporters and ion channels



25	20031023	96	US 2003019894 4 A1	Compositions and methods for reverse transcription of nucleic acid molecules
----	----------	----	--------------------------	--

	Issue Date	Page s	Document ID	Title
26	20031002	96	US 2003018627 0 A1	Compositions and methods for reverse transcription of nucleic acid molecules
27	20030911	121	US 2003017127 5 A1	Transporters and ion channels
28	20030911	31	US 2003017062 2 A1	GENERATION OF SPECIFIC BINDING PARTNERS BINDING TO (POLY) PEPTIDES ENCODED BY GENOMIC DNA FRAGMENTS OR ESTS
29	20030213	97	US 2003003208 6 A1	COMPOSITIONS AND METHODS FOR REVERSE TRANSCRIPTION OF NUCLEIC ACID MOLECULES
30	20021107	37	US 2002016473 6 A1	Ginkgo biloba levopimaradiene synthase
31	20020627	97	US 2002008158 1 A1	COMPOSITIONS AND METHODS FOR REVERSE TRANSCRIPTION OF NUCLEIC ACID MOLECULES
32	20020530	35	US 2002006539 2 A1	METHODS FOR PRODUCTION OF PROTEIN
33	20010823	22	US 2001001631 4 A1	LINKING GENE SEQUENCE TO GENE FUNCTION BY THREE DIMENSIONAL (3D) PROTEIN STRUCTURE DETERMINATION
34	20060124	92	US 6989259 B2	Recombinant methods for making reverse transcriptases and mutants thereof
35	20050920	80	US 6946283 B2	Ginkgo biloba levopimaradiene synthase

36	20041228	90	US 6835561 B1	Composition of reverse transcriptases and mutants thereof
----	----------	----	------------------	--

	Issue Date	Pages	Document ID	Title
37	20040727	112	US 6767719 B1	Mouse telomerase reverse transcriptase
38	20040427	36	US 6727070 B2	Protein/solubility folding assessed by structural complementation
39	20040309	29	US 6703484 B2	Methods for production of proteins
40	20031125	30	US 6653068 B2	Generation of specific binding partners binding to (poly)peptides encoded by genomic DNA fragments or ESTs
41	20030520	42	US 6566108 B1	Expression of functional cytochrome P450 monooxygenase system in enterobacteria
42	20030211	96	US 6518019 B2	Compositions and methods for reverse transcription of nucleic acid molecules
43	19991012	19	US 5965399 A	Cloning and expression of rat liver and porcine liver ribonuclease inhibitor
44	19990803	34	US 5932440 A	Mammalian ribonuclease inhibitors and use thereof
45	19990720	28	US 5925523 A	Intracell trap assay, reagents and uses thereof
46	19931214	26	US 5270179 A	Cloning and expression of T5 DNA polymerase reduced in 3'- to-5' exonuclease activity

	<b>L #</b>	<b>Hits</b>	<b>Search Text</b>
1	L1	1	"5270181".pn.
2	L2	10	inclusion adj partner
3	L3	0	l1 and l2
4	L4	2782 74	dye\$2
5	L5	0	l1 and l4
6	L6	5827	thioredoxin
7	L7	8306 87	clon\$3 or express\$3 or recombinant
8	L8	4203	l6 same l7
9	L9	15	l4 same l8
10	L10	3334 3	CHATTERJEE LONGO FLYNN OBERFELDER
11	L11	210	l6 and l10
12	L12	7526	inclusion adj bod\$3
13	L13	46	l11 and l12